A STUDY ON PREVALENCE OF HEARING LOSS AS A COMPLICATION OF DIABETES

DISSERTATION

SUBMITTED FOR

M.S IN OTO RHINO LARYNGOLOGY

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

DEPARTMENT OF E.N.T

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

PEELAMEDU, COIMBATORE- 641 004

TAMILNADU, INDIA

APRIL 2015

CERTIFICATE

This is to certify that the thesis entitled '**A study on prevalence of hearing loss as a complication of diabetes'** is a bonafide work of **Dr. Prathula Sivakumar** done under the guidance and supervision of **Dr. S. Palaninathan, MS** in the Department of E.N.T, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of Dr. MGR Medical University for the award of M.S. Degree in Oto-Rhino-Laryngology.

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INTRODUCTION

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A study on prevalence of hearing loss as a complication of diabetes

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The 56 organ of Corti is situated on the basilar membrane. It is the sense organ of hearing and balance. The organ of Corti contains the tunnel of Corti , hair cells , supporting cells , tectorial membrane. The inner and outer rods form the tunnel of corti. The fluid inside is called the cortilymph. The hair cells act as a significant receptor cells for hearing. It transforms sound to electrical energy. 51 Inner hair cells are arranged in a single row while the outer hair cells are placed in 3 to 4 rows. The inner hair cells are richly supplied by afferent cochlear fibres and it is probably more important in transmission of auditory impulses. The outer hair cells mostly receive efferent innervations from the Olivary complex. It is concerned with modulating the function of the inner hair cells. There are a total of 3500 inner hair cells and they are flask shaped . The outer hair cells are 12000 in number and they are cylindrical.

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March 01, 2013

To
Dr Prathula Sivakumar
Postgraduate
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The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 8th February, 2013 in its expedited review meeting held at College Council Room, PSG IMS&R, between 2.30 pm and 4.30 pm, and discussed your application to conduct the study entitled:

"A study on prevalence of hearing loss as a complication of diabetes"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Consent forms in English and Tamil
4. Budget
5. Data Collection Tool
6. CV

After due consideration, the Committee has decided to approve the above study.

The members who attended the meeting, at which your proposal was discussed, are listed below:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member - Secretary	Female	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes

The approval is valid for one year.

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee
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We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

Non-adherence to the Standard Operating Procedures (SOP) of the Institutional Human Ethics Committee (IHEC) and national and international ethical guidelines shall result in withdrawal of approval (suspension or termination of the study). SOP will be revised from time to time and revisions are applicable prospectively to ongoing studies approved prior to such revisions.

Yours truly,

Dr S Bhuvaneshwari
Member - Secretary
Institutional Human Ethics Committee

SECRETARY
PSG IMS & R
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INSTITUTIONAL HUMAN ETHICS COMMITTEE

DECLARATION

I hereby decalre that this study dissertation entitled "**A study on prevalence of hearing loss as a complication of diabetes"** was prepared by me under the direct guidance and supervision of Professor of E.N.T, Dr.S. Palaninathan, MS, PSG Institute of Medical Sciences & Research, Coimbatore.

This dissertation is submitted to the Tamil Nadu Dr. MGR Medical University in fulfillment of the University regulations for the award of M.S. degree in Oto-Rhino-Laryngology. This dissertation has not been submitted for the award of any other Degree or Diploma.

Dr. Prathula Sivakumar

CERTIFICATE BY THE GUIDE

This is to certify that the thesis entitled "**A study on prevalence of hearing loss as a complication of diabetes"** is a bonafide work of Dr. Prathula Sivakumar done under my direct guidance and supervision in the Department of E.N.T, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of DR. MGR Medical University for the award of M.S. degree in Oto-Rhino-Laryngology.

Dr. S. PALANINATHAN

Professor

Dept. of. E.N.T

ACKNOWLEDGEMENTS

It is my greatest pleasures to recall the people who have helped me in the completion of my dissertation.

It gives me immense pleasure to express my heartfelt gratitude and sincere thanks to my beloved teacher, Professor and HOD **Dr. George Zacharias**, Dept. of E.N.T, PSG IMS & R, Coimbatore for being an inspiration and for providing all support during this study, without whose towering presence this study would not have been possible.

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I take this opportunity to thank **Dr. Anil Mathew**, Professor in Biostatistics whose inputs were very valuable in completion of this study.

I take this opportunity to recognize the efforts by E.N.T department staff, **Mrs. Chitrapriya, Mrs. Gomathi , Mrs. Anandhi and Ms. Nithya** for helping me collect all necessary patient details. I also want to mention a special thanks to the audiology technicians **Mrs. Prema and Mr. keerthiraj** for helping me in the audiology session.

I am thankful to my family especially my father and my husband who were the real source of encouragement for me during my dissertation work. I thank my post Graduate colleagues for their whole-hearted support.

At the outset I would like to sincerely thank **Dr. S. Ramalingam**, Principal and the Ethics Committee, PSG IMS & R for having consented to this study.

Finally my heartfelt appreciation & greatest thanks to all the **patients** enrolled in this study.

CONTENTS

INTRODUCTION

Hearing impairment is the most common sensory deficit in human beings. It affects more than 250 million people in the world. The consequences of hearing impairment include difficulty in interpret of speech, often producing a decreased ability to communicate, delay in language acquisition, educational and economic drawbacks, social isolation and stigmatization. It may turn bad along with by medical conditions such as hypothyroidism, diabetes, and hyperlipidemia, among others.

Hearing loss can be classified into 3 types:

A. Conductive hearing loss
B. Sensorineural hearing loss
C. Mixed hearing loss

Hearing of an individual could by assessed by clinical and audiometric tests. Pure tone audiometery is a simple audiometric test which is being used to measure the degree of hearing impairment.

Diabetes mellitus is an increasing health problem worldwide and the prevalence is steadily increasing. And it is more pronounced in India (an estimate total of about 40.9 million).

Diabetes Mellitus is a metabolic disorder. It presents because of partial or complete reduction of insulin levels which results in increased levels of blood glucose in association with long term complications which may be vascular or neurological. Among the metabolism disorders of glucose, diabetes is found commonly related to auditory disorders. It also affects metabolism of lipids and proteins. Though Diabetes Mellitus has various etiologies, the most common presentation is hyperglycemia. The physiological basis of type 2 diabetes mellitus is a combined form of both impairment of beta cell function, with significant raise in peripheral levels of insulin resistance near the levels of receptor and post receptor. The complex arrangement of inner ear makes it potential target of hyperglycaemic damage.

Diabetic patients are more prone to complications of hyperglycemia, as all body cells are exposed to high levels of plasma glucose. The organ of corti cells are important structures for hearing mechanism and turn out to be the potential target for damage, due to high glycemic levels, micro vascular compromise, their complex structure and arrangement. Thus screening of these patients at a higher risk of developing sensory neural hearing loss, will aid in early diagnosis and management.

AIM OF STUDY

1. To Study the prevalence of Hearing loss and its association with Diabetes.

2. To assess the hearing loss in diabetics and correlate it with age and duration of diabetes

REVIEW OF LITERATURE

The pure tone hearing average in frequencies 250, 500, 1000, 2000, 4000, 6000, 8000 Hz heard in more than 25 decibel in the ear is called as hearing loss. Hearing loss is a dysfunction of hearing. Its severity varies from mild; moderate; severe or profound. In general hearing loss can be divided into conductive type, sensorineural type or mixed type.[1]

Hearing impairment is the most common sensory deficit in human populations, affecting more than 250 million people in the world. The consequences of hearing impairment include inability to interpret speech sounds, often producing a reduced ability to communicate, delay in language acquisition, economic and educational disadvantage, social isolation and stigmatization. It may be worsened by some medical conditions such as diabetes, hypothyroidism and possibly hyperlipidemia, among others.[2]

Global Burden of Disease 2000 study which was published in the World Health Report 2001 the adult-onset hearing loss is the 2^{nd} most leading cause of Years lead with diseases in global level, which accounts for 4.6% of total global YLDs [3].

ANATOMICAL CONSIDERATIONS:

The human ear has three parts the outer, the middle and the inner ear. During the 6th week of embryonic life, about 6 tubercles appear near the 1st branchial cleft. These tuberceles gradually coincide to become auricle. By 20th week, pinna achieves adult shape.

The external auditory canal forms from the 1st branchial cleft. In the 16 th embryonic week, the cells multiply from below ectodermal cleft and forms the meatal plug. Recanalisation from this forms the epithelial lining in the bony meatus. Recanalisation starts deep near the tympanic membrane and grows outwards. External ear canal is completely formed in the 28th week.

The development of tympanic membrane is from the three germinal layers. The outer epithelial layer develops from the ectoderm, the inner mucosal layer is from the endoderm and the middle fibrous layer is from the mesoderm.

The Eustachian tube, tympanic cavity, attic, antrum and mastoid air cells forms from the endoderm in the tubotympanic recess. This is found arising from the first and second pharyngeal pouch partially. The malleus and incus develops from mesoderm of the 1st arch whereas stapes develops from the 2^{nd} arch except the footplate and the annular ligament which is formed from the Otic capsule.

The inner ear is the first organ of special senses to be formed in man. The evolution of the inner ear starts during the 3^{rd} week of embryonic life and is completely from the sixteenth week. Ectoderm in this region of the hind brain becomes thick to form the auditory placode which invaginates to become the Auditory vesicle or the Otocyst. This then differentiates to form the endolymphatic duct and sac, the semicircular ducts, saccule, the utricle and the cochlea. The cochlea is fully developed by 20^{th} week of gestation.

The external ear consists of the auricle or pinna, external acoustic canal and tympanic membrane. The entire pinna, except the lobule, and the outer part of external acoustic meatus are derived from a single framework of yellow elastic cartilage which is covered by skin. This is attached tightly to the perichondrium over the lateral surface and it is loosely attached over the medial surface. The external acoustic canal starts below the concha till the tympanic membrane. It measures around 24mm in its posterior wall. There are two parts of the canal namely the cartilaginous and the bony part. The cartilaginous part measures about 8mm and is the outer part of the canal. The skin, covering this region is thicker and it contains ceruminous glands and pilosebaceous glands. The hair follicles are limited only in the outer part of canal. The bony part forms inner two- thirds and measures 16mm. Skin lining this region is thiner and it continuity with the tympanic membrane. It is free of hair follicles and cerumin containing gland.

The tympanic membrane forms the division from the external canal and the middle ear. They are placed obliquity, measures about 9- 10mm in height. It is divided into two parts,the pars tensa and the pars flaccida. Pars tensa forms most of the tympanic memebrane . The central part is withdrawn inwards near the tip of the malleus and this region is called Umbo. Bright cone of light is usually found radiating from the tip of malleus extending till the periphery of the anteroinferior quadrant. Pars flaccid also known as Sharpnel's membrane is situated above the lateral process of malleus between the notch of Rivinus and the anterior and posterior malleal folds. It appears pink and not so taut. The tympanic membrane is divided into 3 layers (i) The epithelial layer is outer most, found in continuity with the meatus skin lining (ii) The mucosal layer is innermost, continous from the mucosa in the middle ear. (iii) Fibrous layer is in middle, encloses the handle of malleus. This layer has 3 types of fibers, namely the radial, circular and parabolic fibers.

The middle ear along with eustachian tube, aditus, antrum and mastoid air cells is called the middle ear cleft. It is covered by mucosa and it is filled with air. Middle ear is compared to a box with six sides with a roof, a floor, lateral, medial, anterior and posterior walls. The mastoid antrum is the largest cell and it contains air and communicates with the attic through the aditus in the upper part. The tegmen antri forms the roof and this separates it from the middle cranial fossa. Aditus is an opening through which the attic communicates with the antrum. The facial nerve is found just below the aditus

Three ossicles are found in the middle ear. They are the malleus, the incus and the stapes. Malleus has a head, handle, neck, a lateral process and anterior process. The head and neck of malleus lie in the attic. The lateral process forms a knob- like projection and gives attachment to anterior and posterior malleal folds. The incus consists of the body and short process both of which lie in the attic. The long process is attached to the head of stapes. The stapes is divided into the head, foot plate, neck, anterior and posterior crura. It is attaches to the oval window by the annular ligament. The ossicles direct the sound energy from the tympanic membrane into the oval window and then to the inner ear.

The internal ear also called the labyrinth is an organ of importance for balance and hearing. It contains the bony and membranous labyrinth. Membranous labyrinth contains fluid which is clear called endolymph. Perilymph is seen in the space seperating the membranous and bony labyrinth . The bony labyrinth has three parts: the vestibule, the semicircular canals and the cochlea. The membranous labyrinth consists of the cochlear duct, the utricle, saccule, the endolymphatic duct and sac and three semicircular ducts,

The organ of Corti is situated on the basilar membrane. It is the sense organ of hearing and balance. The organ of Corti contains the tunnel of Corti , hair cells , supporting cells , tectorial membrane . The inner and outer rods form the tunnel of corti. The fluid inside is called the cortilymph. The hair cells act as a significant receptor cells for hearing. It transforms sound to electrical energy. Inner hair cells are arranged in a single row while the outer hair cells are placed in 3 to 4 rows. The inner hair cells are richly supplied by afferent cochlear fibres and it is probably more important in transmission of auditory impulses. The outer hair cells mostly receive efferent innervations from the Olivary complex. It is concerned with modulating the function of the inner hair cells. There are a total of 3500 inner hair cells and they are flask shaped . The outer hair cells are 12000 in number and they are cylindrical.

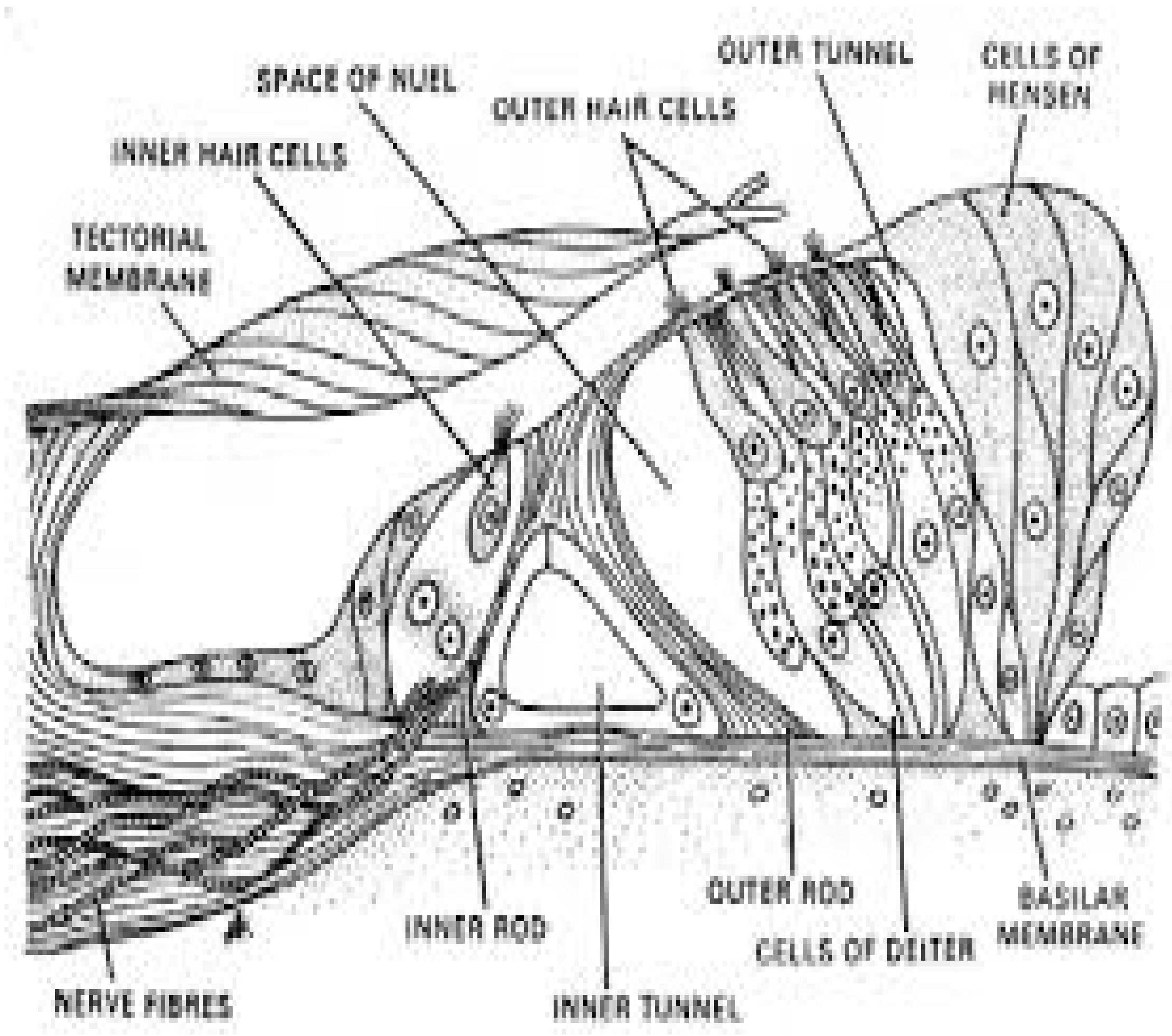


Figure 1: Organ of corti

Acoustics:

Sound is a form of energy produced by a vibrating object. A sound wave consists of compression and rarefaction of molecules of the medium in which it travels. The sound velocity differs from media to media. Sound travels fastest in solid medium.

Frequency is the number of cycles per second. The unit of frequency is Hertz (Hz) named after Heinrich Rudolf Hertz who was a German scientist. A sound of 1000 Hz means 1000 cycles per second.

Pure tone is a single frequency of sound. In pure-tone audiometry the threshold of hearing is measured in decibles for varied pure tones ranging from 125 to 8000.

Complex sound is a sound which contains many frequency. Human voice is an example of complex sound.

Pitch is a subjective sensation which is produced by frequency of sound. More the frequency, more is the pitch.

A complex sound consists of basic frequency, i.e. the lowest frequency in which a source is set into vibrations. Frequencies above this level are termed as overtones. The latter determines the quality or the timber of sound.

Intensity is the sound strength which determines its loud nature. It is usually calculated in decibles. At an approximate distance of 1 metre, the intensity of

Whisper = 30 dB

Normal conversation = 60 dB

Shout = 90 dB

Discomfort of the ear = 120 dB

Pain in the ear = 130 dB

Loudness is a sensation that is subjective, which is produced by intensity. More the intensity of sound, more is the loudness.

Decibel(dB) is denoted as 1/10th of a bel and it is named in remembrance of Sir Alexander Graham Bell. It is the logarithmic ratio among two sounds which is the sound being detailed and sound of reference. Sound can be measured as power. Level of sound pressure is the measure of sound in audiology. SPL is compared in reference of sound which containd an SPL of 0.0002 dynes/cm2 or 20µ Pa (micropascals) this approximately corresponds to the threshold in normal subjects with hearing under normal limits at 1000 Hz. Decibel is used to avoid large figures of sound pressure level.

$$\text{Sound in dB} = 10 \log \frac{\text{Power of S1}}{\text{Power of S0}}$$

Noise is descirbed as an aperiodic complex of sound. The three varities of noise are:

(a) White noise: this consists of all the frequencies in the entire audible spectrum. It can be compared to white light which contains all the colours in the visible spectrum. This is a broad-band noise generally used for purpose of masking.

(b) Narrow band noise: This is white noise that contains specific frequencies which are above and below the given noise which is filtered out. Thus its frequency range is smaller than that of broad-band white noise. This is also used in masking specific test frequency during pure tone audiometry.

(c) Speech noise: All noises in speech range frequencies are called speech noise (300-3000 Hz). All the other frequencies are generally filtered out.

Masking is a mechanism to produce inaudibility of one sound by the producing another. In clinical audiometry, one ear is occupied by a sound while the other ear is being tested. Masking of ear which is not tested is very essential in all forms of bone conduction tests, while in case of air conduction tests, it is required only when the difference of hearing between two ears exceeds more than 40 dB.

In clinical audiometry, one ear is occupied by a sound while the other ear is being tested. Masking of ear which is not tested is very essential in all forms of bone conduction tests, while in case of air conduction tests, it is required only when the difference of hearing between two ears exceeds more than 40 dB.

Wide range of human voices falls in this range. PTA is the average threshold measure of all three frequencies of hearing. This roughly equal to the speech reception threshold.

Hearing level is the sound pressure level which is delivered by an audiometer at a respective frequency. It is measured in decibels. The reference is maintained at audiometric zero. If an audiometer produces a sound at 70dB, it is represented as 70dB HL.

Physiology of hearing:

Sound waves travel through external auditory meatus and produce vibrations in the tympanic membrane. Vibrations from tympanic membrane travel through malleus and incus and reach the stapes resulting in the movement of stapes. Movements of stapes produce vibrations in the fluid of cochlea. These vibrations stimulate the hair cells in the organ of Corti. This in turn, causes generation of action potential in the auditory nerve fibers. When auditory impulses reach the cerebral cortex, the perception of hearing occurs. Thus, during the process of hearing, ear converts enery of sound waves into action potentials in the auditory nerve fibers. This process is called sound transduction.

Role of inner ear:

Traveling wave: Movement of foot plate of stapes against oval window causes movement of perilymph in scala vestibuli. This fluid does not move all the way from oval window to round window through helicotrema. It immediately hits the vestibular membrane near oval window. This causes movement of fluid in scala media, since the vestibular membrane is flexible. This causes bulging of the basilar membrane towards scala tympani. This increases the elastic tension in basilar fibres in the portion of the basilar membrane. This tension initiates a wave,which travels along basilar membrane towards the helicotrema.

Resonance point: It is a part of basilar membrane, which is activated by traveling wave. Initially each wave is weak. When it travels through the basilar membrane from base towards apex, the wave becomes stronger and at one point it becomes very strong and activates the basilar membrane. This resonance point of the basilar membrane immediately vibrates back and forth. The traveling wave stops here. Distance between stapes and resonance point is inversely propotional to frequency of sound waves reaching the ear. Wave generated by high pitched sound disappears near the base of the chochlea, medium-pitched sound reaches half of the way and wave generated by low pitched sounds travel the entire distance of the basilar membrane.

Excitation of hair cells:

Stereocilia of hair cells in organ of cortii are embedded in tectorial membrane. Haircells are tightly fixed by cuticular lamina reticularis and the pillar cells. When travelling wave causes vibration of basilar membrane at the resonance point, the basilar fiber, pillar cells, hair cells and lamina reticularis move as a single unit. It causes movements of stereocilia leading to excitement of hair cells and generation of receptor potential.

Sound Transduction:

It is type of sensory transduction in the hair cells in the organ of Corti by which sound energy is converted into action potentials in the auditory nerve fiber. Three types of electrical events that occur during sound transduction are :

(1) Receptor potential or chochlear microphonic potential

(2) Endochochlear potential or endolymphatic potential

(3) Action potential in auditory nerve fiber

Role of hair cells:

Inner hair cells and outer hair cells have different roles during sound transduction. The inner haircells are responsible for sound transduction, i.e. these receptor cells are the primary sensory cells, which causes the generation of action potential in auditory nerve fibers. Outer hair cells have a different action. These hair cells are shortened during depolarization and lengthened during hyperpolarisation. This process is called electromotility. This action of outer hair cells facilitates the movement of basilar membrane and increases the amplitude and the sharpness of sound. Hence, the outer hair cells are collectively called chochlear amplifier.

Role of efferent nerve fibers of hair cells:

They play an important role during sound transduction by releasing acetyl choline. Efferent nerve fiber to inner hair cell terminates on the auditory(afferent) nerve fiber where it leaves the inner hair cell. It controls the generation of action potential in auditory nerve fibre by inhibiting the release of glutamate from inner hair cells.

Theories of hearing:

(1) **Telephone theory:** It was postulated by Sir Rutherford in 1880. It is also called frequency theory. According to this theory, the chochlea plays a simple role of a telephone transmitter. In

telephone, sound vibrations are converted into electrical impulses, which are transmitted by cables to the receiving end. Where electrical impulses are converted to sound waves. Similarly, chochlea converts sound waves into electrical impulses of same frequency. Impulses are transmitted by auditory nerve fibres to cerebral cortex, where perception and analysis of sound are done. It is approximated that, the nerve fibres can transmit maximum of thousand impulses per second. Thus,the telephone theory fails to explain the transmission of sound waves with frequency above 1000 cycles per second.

(2) **Volley theory:** Wever postulated this theory in 1949. According to this theory, the impulses of sound waves with frequency above 1000 cycles per second are transmitted by different group of nerve fibers. However there was no evidence to prove it. Thus not accepted by many.

(3) **Resonance theory of Helmholtz:** This theory was proposed by Helmholtz in 1863. According to this theory analysis of sound frequency is the function of chochlea. Helmholtz named the basilar fibers as resonators and compared them with resonators of piano. When a string in piano is struck, sound with a particular note is produced. Similarly, when the sound with particular frequency is

applied , the basilar fibes in a particular portion of the basilar membrane are stimulated.

(4) **Place theory:** According to this theory, nerve fibers from different portions of organ of cortii on basilar membrane give response to sounds of different frequency. Accordingly, corresponding nerve fiber from organ of corti send information to the brain regarding the portion of organ of corti that is stimulated. Many evidences are present to support the place theory. E.g. If a person is exposed to loud noise of a particular frequency for a long period, he becomes deaf for that frequency. It is found that the specific portion of organ of corti is destroyed.

(5) **Traveling wave theory:** This theory was derived from place theory. It explains the travelling wave generation in basilar membrane.

Auditory Pathway:

Hair cells in the organ of corti are the receptors of auditory sensation. All hair cells are innervated by afferent and efferent nerve fibers. The afferent cells forms the auditory pathway. First order neurons of auditory pathway are the bipolar cells of spiral ganglion. Their long processes leave the ear as cochlear nerve fibers and enter medulla oblongata, where they divide into dorsal and ventral cochlear nucleus on same side of medulla.

These act as second order neurons, where they run as different groups to cross the superior olivary nucleus, lateral lemniscus and recticular formation. The third order neurons are in the superior olivary nuclei and nucleus of lateral lemniscus. Fibers from medial geniculate body go the tremporal cortex, via the internal capsule as auditory radiation. Some fibers run to inferior colliculus which are responsible for reflex movements of head in response to auditory stimuli. The cortical auditory centers in the temporal lobe of cerebral cortex lie are the primary auditory area 41; 42 and Wernicke area. The secondary auditory area is 22.

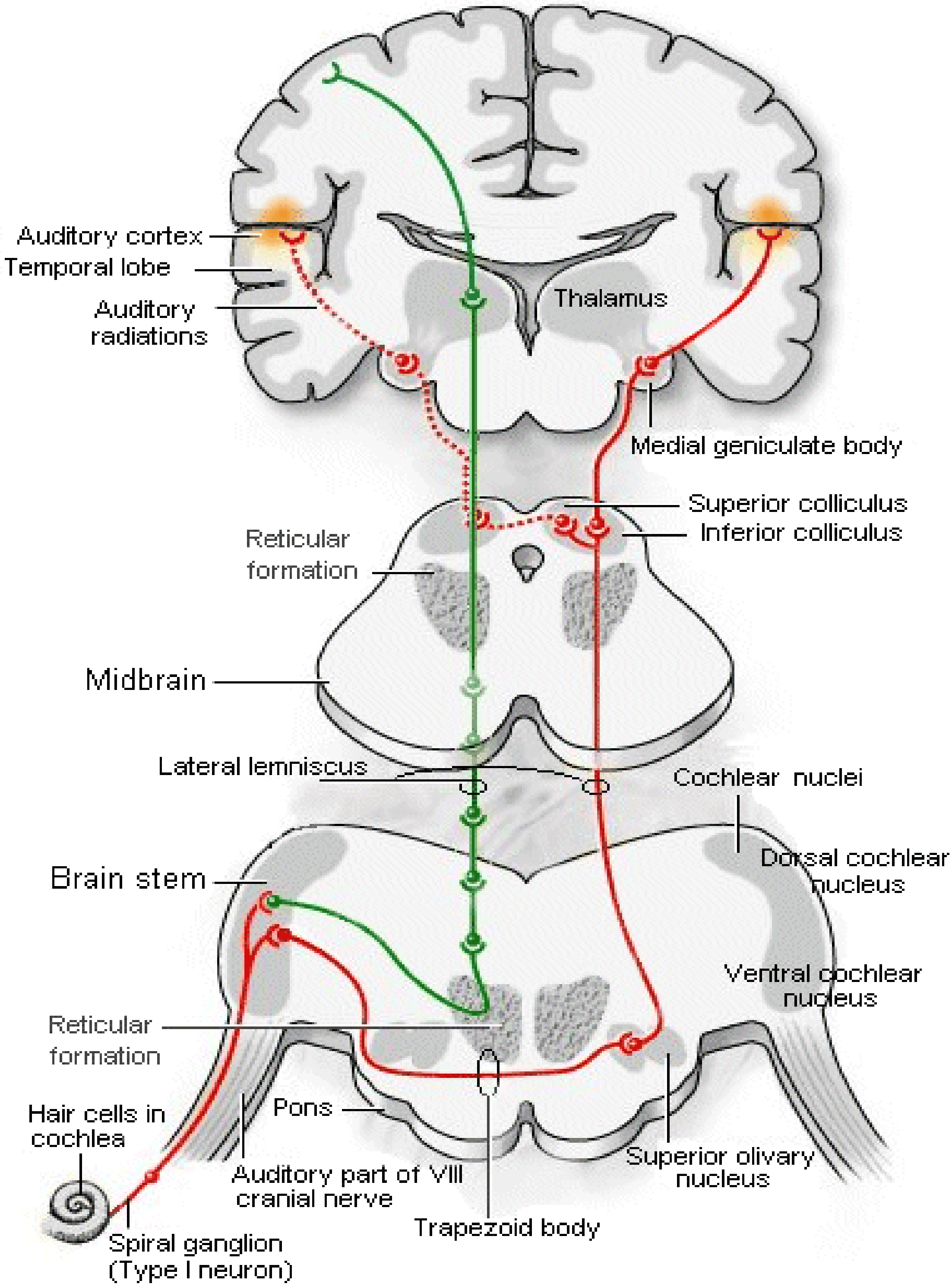


Figure. 2. Auditory Pathway

Hearing loss:

Hearing loss can be of three types (i) Conductive hearing loss (ii) Sensorineural hearing loss (iii) Mixed hearing loss. While auditory function is being assessed it is important to find out the type of hearing loss; degree of hearing loss; site of lesion and cause of hearing loss. Hearing of an individual can be tested by clinical and audiometric tests.

Sensorineural hearing loss: This results from cochlear lesions, VIII th nerve or central auditory pathways. It may be congenital or acquired. The characteristics of sensorineural hearing loss are:

- A positive Rinne test
- Weber lateralized to better ear
- Bone conduction reduced on Schwabach and absolute bone conduction test.
- Mostly high frequency loss
- No gap between air and bone conduction curve on audiometry
- Loss may exceed 60dB
- Speech discrimination is poor
- There is difficulty in hearing when noise is present

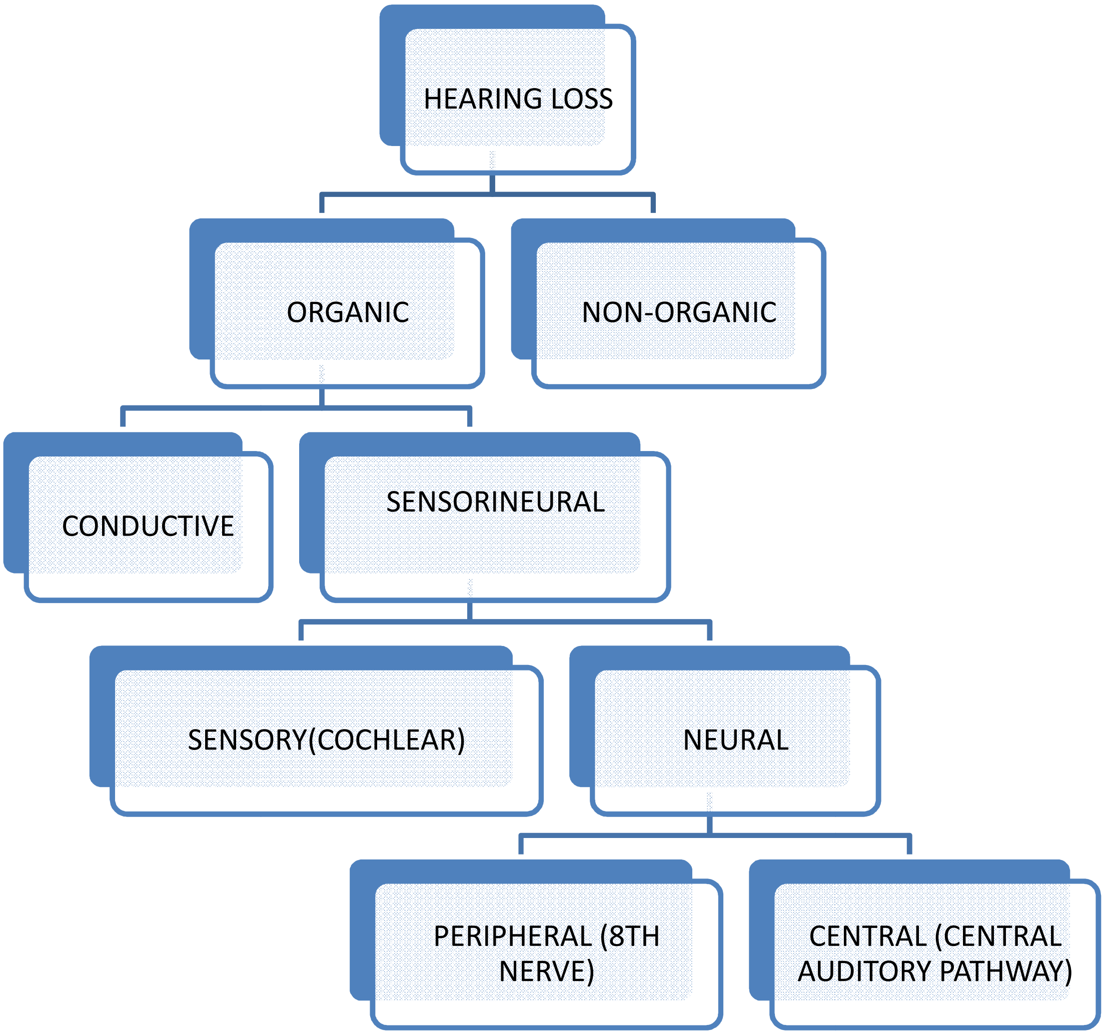


Table: 1: Classification of hearing loss

Congenital sensorineural hearing loss is present from birth and is the result of anomalies of the inner ear or damage to the hearing apparatus by prenatal or perinatal factors.

Acquired sensorineural hearing loss appears later in life. The cause may be genetic or non- genetic. The genetic cause of hearing loss may presents late and damages only the hearing. It can even be a component of a larger syndrome with other the body systems being involved. Common causes of acquired sensorineural hearing loss are:

- Infections of labyrinth
- Trauma to labyrinth
- Noise-induced hearing loss
- Ototoxic drugs
- Presbycusis
- Menier’s disease
- Acoustic neuroma
- Sudden hearing loss
- Systemic disorders like diabetes, hypothyroidsm, kidney disease and autoimmune disorders.

Specific forms of hearing loss:

A. Inflammation of the labrynth

1. Viral labrynthitis : viruses usually reach the inner ear by blood stream affecting stria vascularis , endolymph and organ of corti. Measels,mumps and cytomegalovirus are known to cause labrynthitis.

2. Bacterial : these infections reach the labrynth through the middle year or through CSF.sensory neural deafness following meningitis is a known complication .

3. Bacteria can invade the labrynth along nerves , vessels, coclear aqueduct or endo lymphatic sac and this causes complete distruction of the membraneous labrynth.

4. Syphilitic : sensory neural hearing loss is caused both by congenital and acquired syphilis . syphilitic involvement of the inner ear can cause sudden sensorineural hearing loss which may be unilateral or bilateral. Menier’s syndrome with episodic hearing loss, tinnitus and aural fullness. Tullio phenomenon where loud sounds produce vertigo.

B. Familial Progressive Sensorineural hearing loss: It is genetic disorder characterized by progressive degeneration of cochlea. It may start late in childhood or in early adult life.

C. Ototoxicity: Various chemicals and drugs affect the inner ear and produces sensorineural hearing loss and tinnitus. Symptoms of ototoxicity-hearing loss, tinnitus or giddiness may be noted during treatment or even after completion of entire treatment.

Table 2 : The list of ototoxic drugs are

A. Aminoglycoside antibiotics:

-Streptomycin
- Tobramycin
- Gentamicin
- Neomycin
- Amikacin

B. Diuretics

- Furosemide
- Ethacrynic acid

C. Antimalarials:

- Quinine
- Chloroquine

D. Cytotoxic drugs:

- Nitrogen mustard
- Cisplatin
- Carboplatin

E. Analgesics:

- Ibuprofen
- Salicylates
- Indomethacin
- Phenyl butazone

F. Chemicals:

-Alcohol
- Tobacco
- Carbon monoxide poisoning

G. Miscellaneous:

- Erythromycin
- Ampicillin
- Propranolol

D. Noise trauma:

Hearing loss due to exposure to noise exposure is known in boiler makers, iron smiths, copper smith and artillery men. Now a day's noise trauma has become very significant as it has become an occupational hazard. Hearing loss caused by excessive noise can be divided into two groups:

1. **Acoustic trauma:** Permanent damage to hearing can occur even on single brief exposure to very intense sound. E.g.: Gunfire; explosion. Sudden loud sounds damage cause damage to the outer hair cells disrupting the organ of corti and will result in rupture of the Reissner's membrane. In some cases rupture of tympanic membrane and disruption of ossicular chain can happen in severe blast.

2. **Noise-induced hearing loss:** Here hearing loss occurs following chronic exposure to less intense sounds. This is mostly noted as occupational hazard in people working in noisy environment.

The damage caused by noise trauma depends on many factors like:

a. Frequency of noise
b. Intensity and duration of noise
c. Continuous or interrupted noise
d. Susceptibility of the individual
e. Any pre-existing ear disease

E. **Sudden Hearing loss:** It is a sensorineural hearing loss, that is developed over a period of hours or few days. Hearing loss may be partial or complete. It is mostly unilateral and may be accompanied by tinnitus or spells of vertigo**.** Mostly the cause of this sudden deafness remains unclear and termed as idiopathic. The three main aetiological factors generally considered are: viral, vascular or rupture of cochlear membrane. The other factors causing sudden hearing loss are:

- Infections
- Trauma
- Vascular
- Ear pathology like Meniere's disease.
- Toxic
- Neoplastic
- Psychogenic

F. **Presbycusis:** Sensorineural haering loss associated with aging physiology in the ear is termed as presbycusis. Generally it manifests at 65 years or even earlier. The four pathological types are:

- Sensory
- Neural
- Strial or metabolic
- Cochlear conductive

The tests for hearing are:

A] Clinical tests of hearing are (i) Finger Friction Test (ii) Watch Test (iii) Speech Tests (iv) Tuning Fork Tests which include (a) Rinne test (b) Weber test (c) Absolute bone conduction test (d) Schwabach's test (e) Bing test (f) Gelle's test.

Rinne tuning fork test: is formulated to find the difference between air conduction with that of bone conduction. In normal conditions, air conduction is more than bone conduction and the tuning fork will be heard loud in the opposite ear canal than when it is placed over the mastoid bone behind.

The alternate method for doing this test is by threshold comparison method. In this method the activated tuning fork is held opposite the external canal till it is no longer heard. It is then placed over the mastoid process. If sound is heard once more, then it is considered that bone conduction is better than air conduction and the test is considered negative. The test when performed with 256 Hz tuning fork has greater sensitivity and specificity. The specificity of this test increases above 30dB conductive deafness, decreases when narrow air-bone gap is found. False positivity of this test is about 20%.

Weber tuning fork test: This test is used only if asymmetrical or unilateral hearing is seen in patients. The basis of this is by placing a tuning fork in the centre of the skull being heard louder in the ear with a conductive impairment in case of sensorineural loss it is louder in the better ear.

This difference can be distinctive only, if the examiner has done a clinical hearing test previously and if he knows which ear has better hearing. The test is done by keeping an activated tuning fork on the forehead or over the bridge of nose or on the incisor teeth. The tuning fork can also be placed over the vertex of the skull in the midline. Then the patient is asked to identify the ear in which the sound is heard or otherwise in which ear the sound is louder. If the sound is heard in the better hearing ear, then the poorer ear is considered to have sensorineural impairment. If it is heard in the affected ear, then it is considered as conductive impairment. This test has a low sensitivity and specificity.

Absolute bone conduction test: Bone conduction is measurement of the function of cochlea. Here bone conduction of the patient is compared with the examiner, assuming the examiners have hearing within normal limits. The external auditory canal of both the examiner and the patient are closed, so that ambient noise entering through air conduction route is prevented. In case of conductive hearing loss, the subject and the examiner hears the tuning fork for equal time period. Whereas in case of sensorineural loss, the subject perceives the tuning fork for a brief period of time.

Schwabach's test: Here again the bone conduction of the patient is compared with the examiner, assuming that he has hearing within normal limits. But in this test, the meatus is not occluded. Schwabach's test is reduced in case of sensorineural deafness and lengthened in case of conductive deafness.

The Bing test: it is done on the same basis of Weber test in which closing of the external auditory canal increases the tuning fork sound in the ear to be tested, if conductive hearing loss is present. It is done by keeping a vibrating tuning fork over the mastoid process and the external auditory meatus is also occluded.

If increase in sound is present then there is less likelihood that there is conductive hearing loss. However, if it remains the same, then it is more likely to be a conductive deafness. The specificity and sensitivity of this test is also very low. Most of the situations normal individuals are identified as conductive deafness. This test is not used widely.

Tuning fork tests should generally be reserved for situations where audiometry is not satisfactory. The results must be interpreted keeping in mind the low sensitivity and specificity.

B] Audiometric tests are (i) Pure tone audiometry (ii) Speech audiometry (iii) Bekesy audiometry (iv) Impedance audiometry.

C] Special tests of hearing are (i) Recruitment (ii) Short increment sensitivity index (SISI) (iii) Threshold tone decay test (iv) Evoked response audiometry (v) Otoacoustic emissions (vi) Central auditory tests (vii) Hearing assessment in children and infants.

Pure tone audiometry : Pure tones are produced by an electronic device called an audiometer. The intensity of these tones can be increased or decreased in steps of 5 dB. Generally threshols for air conduction are calculated for tones of 125, 250, 500, 1000, 2000, 4000 and 8000 Hz and for thresholds of bone conduction, it is done at 250, 500, 1000, 2000 and 4000 Hz.

The measure of intensity of tones that has to be increased more than the level of normal is considered as the degree of hearing impairment in that particular frequency. Audiogram is a graphical representation of the charted values. The bone conduction threshold is a measurement of function of cochlea. The variations in the air conduction and bone conduction thresholds (A-B gap) are a measurement of the degree of conductive hearing loss. It is observed that the calibration of audiometer is done in such a way that the perception in a normal person, both the air and bone conduction remains at zero dB and no A-B gap is noted. The turning fork test generally shows AC>BC.

When the difference of hearing in the 2 ears is 40 dB or more in thresholds of air conduction then the ear with better hearing is masked so as to not to get a shadow curve in the better ear that is not being tested. In the same way masking is important in all studies of bone conduction. Masking is carried out by delivering a narrow-band noise to the ear that is not being tested. The benefits of pure tone audiogram are (i) It is a measurement threshold for hearing in both air and bone conduction, and also the type and degree of hearing loss. (ii) It is a reference for future record. (iii) Hearing aids can be prescribed only after an audiometery (iv) Speech reception thresholds can be predicted with its help (v) The degree of handicap can be assessed for medicolegal issuses.

Figure. 3: Image showing patient undergoing Pure tone audiometry

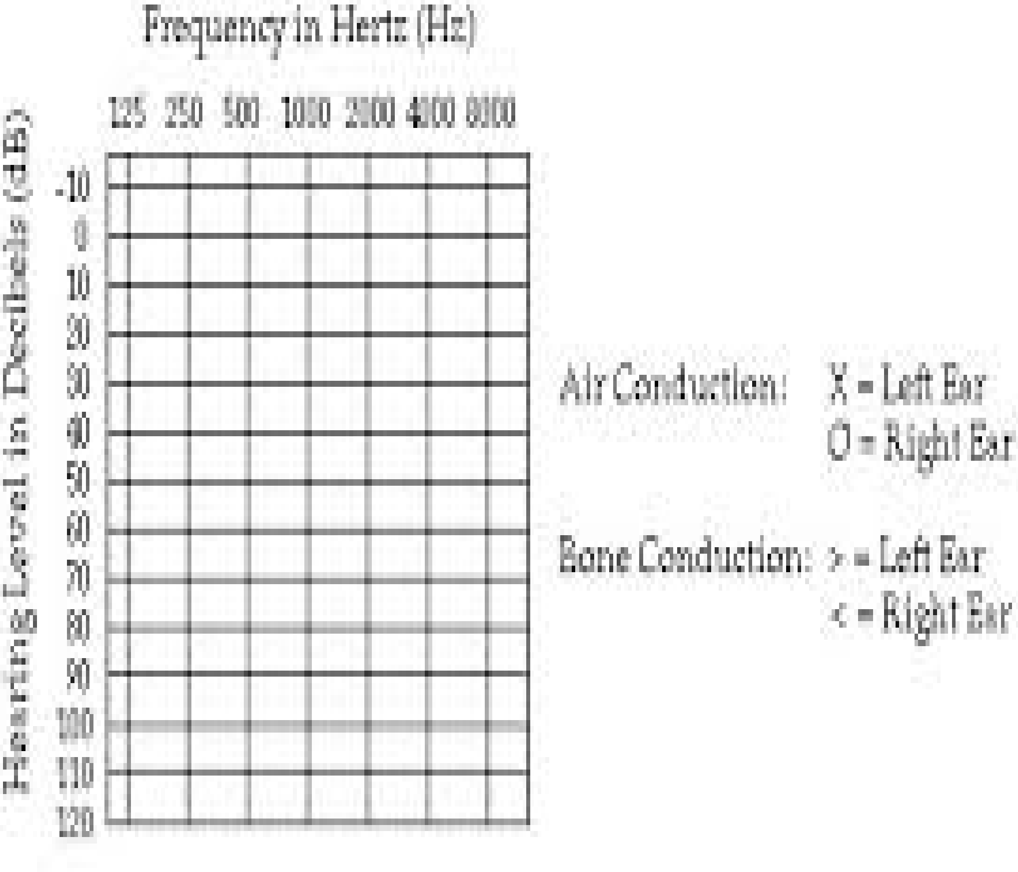


Figure. 4. Audiogram

Equipment:

A pure tone audiometer is designed with a set of basic functions. The technical requirements for the instrument are specified by international standard, in which four types are identified based on complexity of functions and working range of various characteristics.

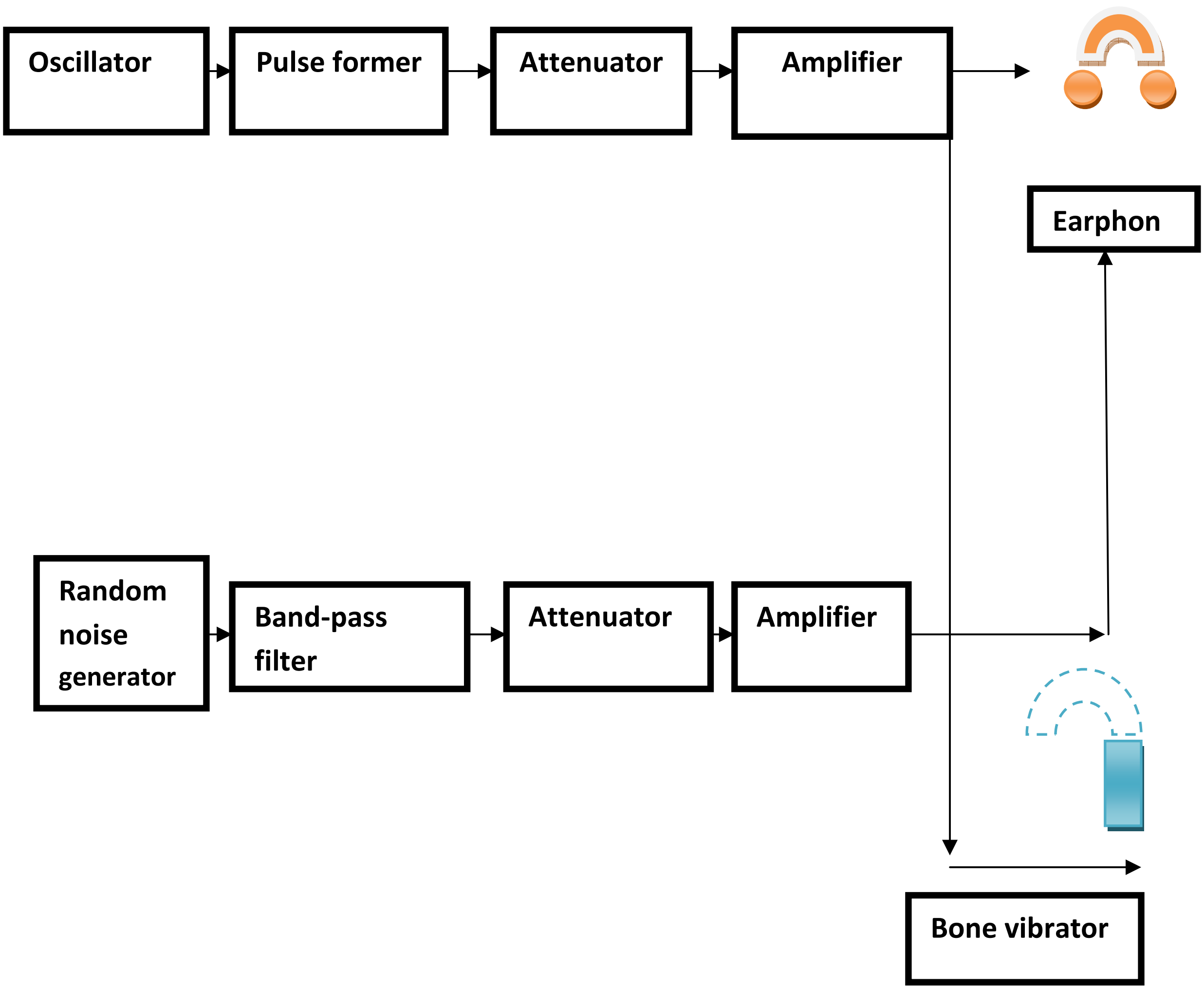


Fig.4: Flow diagram of pure tone audiometery

speech audiometry is a test in which the patient's capacity to hear and to understand speech is calculated. 2 parameters are taken into consideration: (i) Speech reception threshold (ii) The discrimination score. SRT is the intensity at which minimum words of 50% are reciprocated righttly by the patient. The headphones of an audiometer delivers a set of spondee words to both nears.

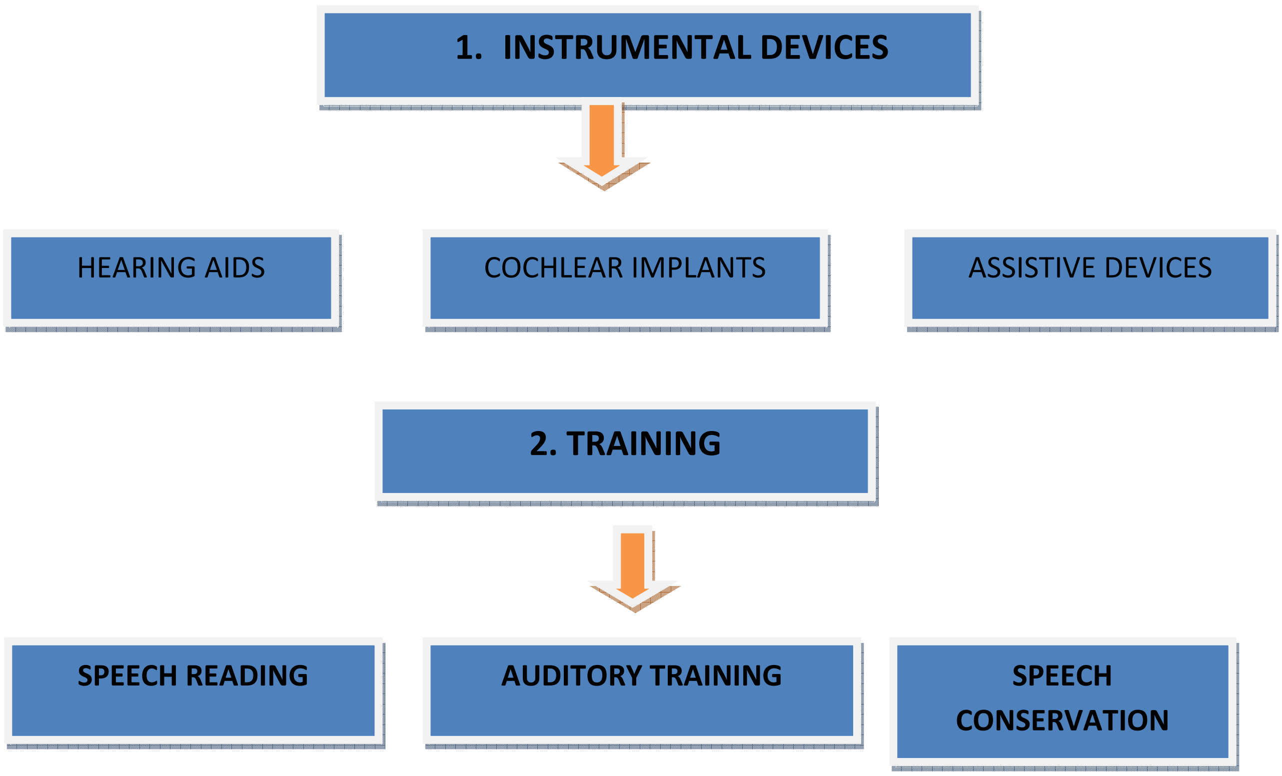
An SRT more than pure tone average of 10dB suggests that hearing loss is of functional type. Discrimination score is also termed as speech recognition score. It is a measure of patient's capacity to understand speech. Here phonetically balanced words are delivered to both ears at an intensity 30-40 dB above the SRT.

Thus, speech audiometry helps (a) to differentiate an organic hearing from the functional one. (b) to indicate at which intensity the discrimination score is best, and this is useful for fitting apt hearing aid and setting its volume for optimal hearing and (c) to differentiate a cochlear from retrocochlear lesion.

MANAGEMENT OF HEARING IMPAIRMENT:

Management of individuals with hearing impairment can be determined by the degree of hearing impairment, whether the impairment is sensorineural, conductive or mixed. Ears with mild, moderate or severe impairment are given the option of hearing aids. Those with profound or total impairment, cochlear implants may be suggested. Middle ear implants were developed for those with conductive hearing loss but in long term it could be used for sensorineural hearing loss.

All individuals with hearing impairment need some rehabilitation for communication. The various means of rehabilitation are:



Hearing aids:

Hearing aids partially overcome the deficits associated with hearing loss. For a sensorineural hearing loss, there are several deficits to overcome. Some sounds are inaudible and some are heard because partial spectra are audible.

Hearing aids are devices that amplify sound by electrical or chemical means. The basic components of a hearing aid are:

- Microphone: They collect sounds and transform it into electrical energy. The microphones are generally magnetic in type. It consists of diaphragm that converts sound energy into mechanical energy.

- Amplifier: The function is to increase the electrical voltage which is received from the microphone. An implanted transmitter in the amplifier increases the voltage.
- Receiver: It transfers electrical energy back into sound waves with a much greater amplitude than those which fell upon the microphone. The receivers are both air conduction type as well as bone conduction type. These magnetic receivers have their diaphragm connected to a vibrator which is placed over the mastoid process. The vibrations are transmitted to the bony labyrinth and fluid in the cochlea.
- Battery : They are placed in the device to supply power.

Diabetes mellitus is characterized by chronic hyperglycemia with disturbances of carbohydrate, protein and fat metabolism resulting from defects in insulin secretion, insulin action, or both. The effects of diabetes mellitus include long term damage, dysfunction and failure of organs like eye, ears, heart, kidneys and blood vessels. It may present with symptoms like thirst, polyuria, polyphagia, blurring of vision and weight loss. And in more severe forms it may present as ketoacidosis or nonketotic hyperosmolarity, which in the absence of treatment leads to stupor, coma and death.

A number of specific causes of diabetes mellitus have been identified, the etiology and pathogenesis is not clear. The majority of cases fall under two broad etiopathological categories, now called as type 1 and type 2 diabetes. Recently the WHO along with expert committee of the American Diabetes Association (ADA) has given a new classification.

Table 4: Etiological classification of disorders of glycemia

TYPE 1
Beta cell destruction usually leading to absolute insulin deficiency
A) Autoimmune
B) Idiopathic
TYPE 2
A) Predominantly insulin resistance
B) Predominantly insulin secretory defects
OTHER SPECIFIC TYPES OF DIABETES
A) Genetic defects of beta cell dysfunction
B) Genetic defects in insulin action e.g: Type A insulin resistance
C) Diseases of exocrine pancreas e.g: Fibro calculus pancreatopathy
D) Endocrinopathies e.g: Acromegaly, cushings etc
E) Drugs or chemical – induced e.g: Glucocorticoids
F) Infections e.g: congenital rubella
G) Uncommon forms of immune-mediated diabetes e.g: Stiff Man Syndrome
H) Other genetic syndromes
GESTATIONAL DIABETES

This classification differs considerably from the previous recommendation which classified using terms such as insulin-dependent diabetes and non-insulin dependent diabetes [4]. These terms were misused and classified patients based on treatment needs rather than etiologic characteristics. So the terms type 1 and type 2 diabetes have been adopted for the most common forms of diabetes mellitus.

Clinical Stages:

Individuals who develop diabetes pass through several clinical stages during the development. Initially, glucose regulation is normal and no abnormality of glycemia can be identified even if the individual undergoes oral glucose tolerance test. This stage is followed by a period of variable duration where in glucose regulation is impaired. Some amount of fasting glucose concentration may be noted or oral glucose test may show impairment.

Once diabetes develops, glycemia may be controlled by lifestyle changes like diet and increased physical activity in some patients, whereas in others oral hypoglycemic agents or insulin is needed for control or prevention against ketosis and ketoacidosis.

If insulin is required to prevent ketosis such patients are labeled as "insulin requiring for survival". In all forms remission may be present.

Patients may revert to having impaired glucose regulation or even normal glycemia, particularly if diabetes is of recent onset. This is seen most frequently in patients with recent-onset type 2 DM in whom lifestyle intervention or early aggressive treatment may result in apparent reversal of the abnormality with reversion to impaired or normal glucose tolerance [5].

This may be seen in type 1 diabetes also, where in a short period of insulin treatment may result in a variable period when insulin is no longer required for survival and glucose tolerance may improve, the so called honeymoon period. Eventually these patients need insulin treatment for survival [6].

All subjects with diabetes can be classified according to clinical stages regardless of underlying etiology of diabetes. The stage of glycemia may change over time depending on the extent of underlying disease process. Impaired glucose regulation refers to the metabolic state intermediate between normal glucose homeostasis and diabetes that can be identified by impaired glucose tolerance or impaired fasting glucose [7]. They are not synonymous and represent different abnormalities of glucose regulation, even if they occur together. These patients are at a higher risk of progressing to diabetes.

ETIOLOGIC TYPES

Type 1 Diabetes Mellitus: It is a form of diabetes primarily due to β- cell destruction. This usually leads to a type of diabetes in which insulin is required for survival. These individuals are metabolically normal before the disease is clinically manifested, but the process of β- cell destruction can be detected earlier by the presence of certain antibodies like anti-GAD, anti-islet cell or anti-insulin antibodies. Individuals with one or more of these antibodies can be subclassified as type 1A, immune mediated type 1 diabetes. Type 1 diabetes can also occur in the absence of autoimmune antibodies and without evidence of any autoimmune disorders. It is a progressive form of disease with marked hyperglycemia resulting in insulin requirement for prevention of ketosis and survival. These individuals are classified as type 1B or idiopathic diabetes[8].

The rate of β- cell destruction is variable, being rapid in some individuals especially in infants and children, and slower in adults. Individuals become dependent on insulin for survival only many years after the detection of diabetes. Type 1 diabetes patients have low or undetectable levels of insulin and plasma C- peptide. These patients are more prone for ketoacidosis, although they have no clinical evidence of autoimmune antibodies. They may suffer from episodic ketoacidosis, but pathogenetic basis for this insulinopenia remains unclear.

Type 2 Diabetes Mellitus: It is the most common form of diabetes. It is characterized by disorders of insulin action and insulin secretion. usually both are present at the time of clinical manifestation.

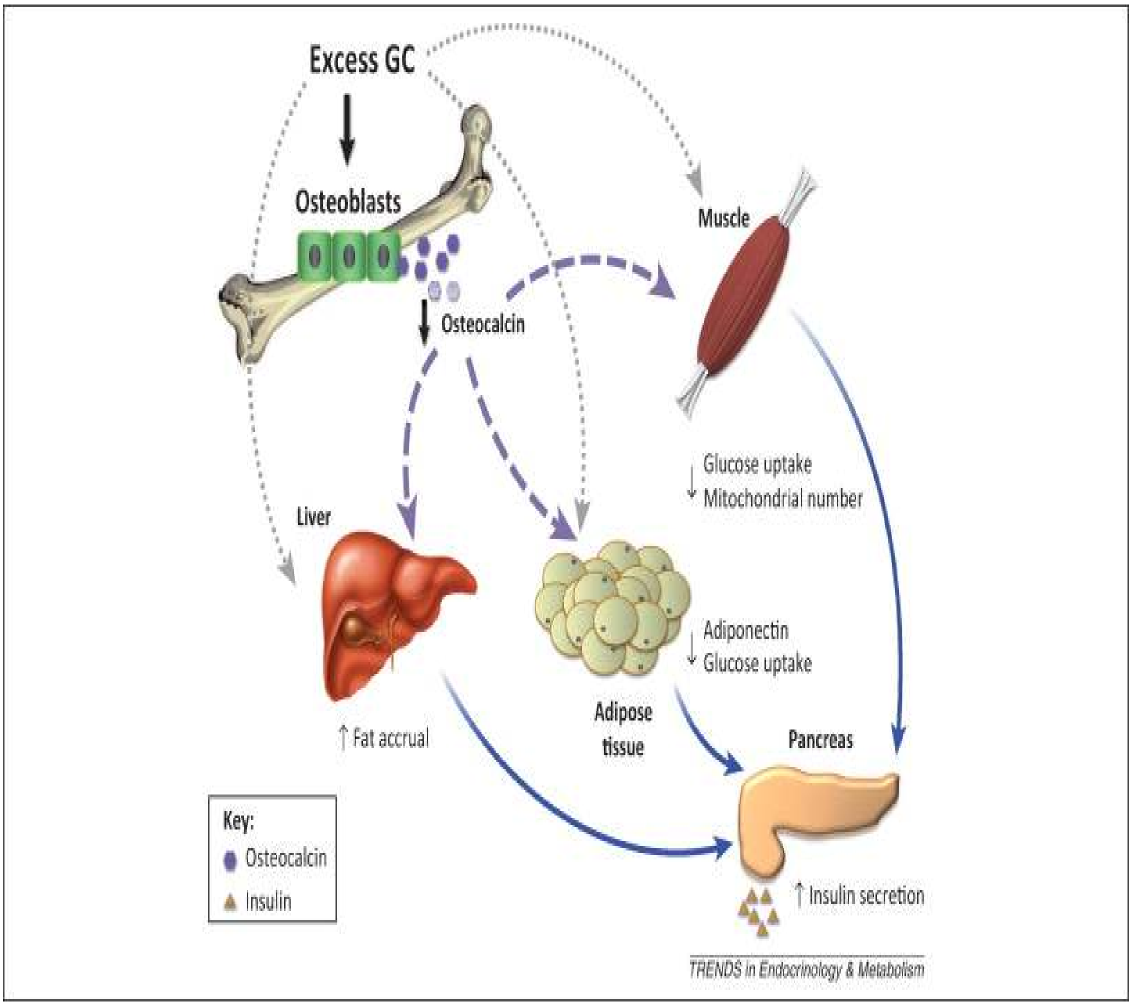


Fig. 5: METABOLISM OF INSULIN PATHWAY

The specific etiology of this form of diabetes is yet unknown. Auto immune destruction of β- cell does not occur in this form of diabetes. Type 2 diabetes patients usually have insulin resistance rather than absolute insulin deficiency. These patients do not need insulin treatment for survival although many may require it for glycemic control. This form of diabetes is associated with progressive β- cell failure with increasing duration of diabetes [9]. Ketoacidosis rarely occurs by itself but conditions associated with stress and other illnesses such as infection can increase the risk

Type-2 diabetes patients are mostly obese when they develop diabetes and obesity aggravates the insulin resistance.this form of diabetes goes undiagnosed for many years because the hyperglycemia develops gradually and in early stages it is not severe enough to produce the classic symptoms of diabetes.however these patients are at increased risk of developing macrovascular and microvascular complications.

The circulating levels of insulin may be normal or elevated yet insufficient to control glucose levels in the blood within the normal range because of insulin resistance. Thus they have relative insulinopenia. Insulin resistance may improve with weight reduction or pharmacologic treatment and results in normalization of glycemia. Women with previous history of gestational diabetes and individuals with characteristics of insulin resistance syndrome like hypertension and dyslipidemia are at higher risk of developing type 2 diabetes. The risk also increases with age, obesity and physical inactivity. Lifestyle measures to reduce weight such as dietary regulation and increased physical activity reduce or delays the development of diabetes [10; 11].

Other Specific Types of Diabetes Mellitus

Maturity-onset diabetes of the young (MODY):

This subgroup includes a relatively rare monogenic disorder characterized by non–insulin dependent diabetes with autosomal dominant inheritance and the age of onset is 25 years or younger. Patients are not obese, and their hyperglycemia is due to impaired glucose-induced secretion of insulin. There are Six types of MODY which have been described. Except that in MODY 2, a glucokinase gene is defective, all other types involve mutations of a nuclear transcription factor that regulates islet gene expression. The enzyme glucokinase is a rate-limiting step in glycolysis and determines the rate of adenosine triphosphate (ATP) production from glucose and insulin secretion.

MODY 2, due to glucokinase mutations, is usually mild, associated with only slight fasting hyperglycemia and few diabetic complications. It responds well to hygienic measures or low doses of oral hypoglycemic agents.

MODY 3, is due to mutations in hepatic nuclear factor 1α and is the most common form. It accounts for two thirds of all MODY cases. The clinical course is of progressive beta cell failure and needs insulin for therapy. Mutations in both alleles of glucokinase present with more severe neonatal diabetes.

Diabetes due to mutant insulin receptors: This is a rare subtype of type 2 diabetes who are not obese. There are less than ten families being described in the past. Since affected individuals were heterozygous, diabetes presentation was mild, and appeared only in middle age. It also showed genetic autosomal dominant transmission. There is no evidence of clinical insulin resistance, and these patients respond well with standard therapy

Defects in insulin receptor genes are found in more than 40 people with diabetes. These individuals have the highest insulin resistance associated with acanthosis nigricans. In very rare instances where the two receptor genes are abnormal , newborns present with leprechaun-like phenotype and do not survive through infancy.

Wolfram syndrome:

It is an autosomal recessive neurodegenerative disorder. It first becomes evident in childhood. It consists of diabetes insipidus, diabetes mellitus, optic atrophy, and deafness. This protein forms a part of the unfolding protein response and this protects the beta cells from endoplasmic reticulum stress and apoptosis during periods of high insulin demand. Diabetes mellitus along with optic atrophy presents in the first decade of life.

Sensorineural deafness and diabetes insipidus starts in the second decade of life in 60–70% of individuals . Ureterohydronephrosis, neurogenic bladder, peripheral neuropathy, cerebellar ataxia and psychiatric illness develops later in many patients.

Fig 6: Acanthosis nigricans in the nape of the neck, with typical dark and velvety appearance

Fig 7: Acanthosis nigricans of the axilla, with typical dark coloration and velvety texture

Autosomal recessive syndromes:

In this form, there is homozygous mutations in a number of pancreatic transcription factors like *NEUROG3, PTF1A,* and *GLI. It* causes neonatal or childhood diabetes. Homozygous *PTF1A* mutations results in absent pancreas and cerebellar atrophy; *NEUROG3* mutations causes severe malabsorption and results in diabetes before puberty. Homozygous mutations in *RFX6* causes the Mitchell-Riley syndrome. It is characterized by the absence of all islet cell types apart from pancreatic polypeptide cells, hypoplasia of the pancreas and gallbladder, and intestinal atresia. The gene *EIF2AK3* helps in controlling the pathway for unfolding protein response. Absence of this leads to inadequate response to stress and accelerated beta cell apoptosis. Patients with mutation in this gene leads to neonatal diabetes, epiphyseal dysplasia, developmental delay, hepatic and renal dysfunction (Wolcott-Rallison syndrome).

Diabetes mellitus secondary to other causes:

Glucose intolerance can be caused by endocrinr tumors secreting growth hormone like catecholamines, glucocorticoids, glucagon or somatostatins. In these incidences there is impairment of peripheral responsiveness to insulin. With increase in levels of hormones hepatic output of glucose becomes a main causative factor. Catecholamines causes decrease in insulin release and becomes an added factor in producing carbohydrate intolerance, somatostatin inhibits insulin secretion and plays as a major factor.

Diabetes predominantly occurs in individuals with underlying defects in insulin secretion and hyperglycemia spontaneously resolves when the hormone excess is resolved. Extreme insulin resistance with acanthosis nigricans is a rare syndrome which affects young woman with androgenic features and also in older woman who have circulating immunoglobulin binds to insulin receptors and this reduces the affinity to insulin. Many medications like calcineurin inhibitors, thiazides, β-blockers, corticosteroids, niacin, atypical antipsychotics are known to produce carbohydrate intolerance or even frank diabetes. These medications act by either decreasing insulin secretion or by increasing insulin resistance or both. Corticosteroids are known to increase insulin resistance Thiazide diuretics and β-blockers moderately increase the risk for diabetes. Treating the hypokalemia produced by these thiazides may reverse the hyperglycemia. Atypical antipsychotics, olanzapine and clozapine, in 63articular have been associated with increased risk of glucose intolerance. These medications also causes weight gain and insulin resistance. An increase in rate of developing diabetic ketoacidosis has also been reported.

DIAGNOSTIC CRITERIA FOR DIABETES:

Patients having symptoms such as thirst, polyuria, unexplained weight loss, drowsiness or coma with marked glucosuria, the diagnosis of diabetes can be established by demonstrating fasting hyperglycemia. If the fasting glucose concentration lies in the diagnostic range for diabetes, an oral glucose tolerance test is not required for diagnosis.

A confirmatory test has to be performed because diagnosis of diabetes carries a considerable consequence for the patient. Intraindividual variation or incomplete fasting may result in wrong diagnosis. Whereas if the patient is asymptomatic or has minimal symptoms with fasting or plasma concentration values which are not diagnostic, an oral glucose tolerance test is required to confirm or exclude the diagnosis of diabetes.

Normal glucose tolerance cannot be established on basis of fasting glucose determination alone. In normal healthy individuals, fasting glucose levels should be less than 100mg/dl in venous or capillary plasma and 90mg dl or less in whole blood. Individuals with fasting glucose levels above the characteristic value for normal healthy subjects but below diagnostic value of diabetes have a high likelihood of having either diabetes or impaired glucose tolerance. These levels represent a primary indication for an oral glucose tolerance test to confirm or exclude the diagnosis of diabetes.

Table 5: Diagnostic Criteria

Diagnostic Criteria for Diabetes Mellitus & Related Stages of Glycemia		
Glucose concentration, mg/dl		
	Capillary whole blood	**Venous Plasma**
Diabetes mellitus Fasting Or 2-hour postglucose	>=110 >=200	>=126 >=200
Impaired glucose tolerance Fasting Or 2-hour postglucose	<110 140-199	<126 140-199
Impaired fasting glycemia Fasting Or 2-hour postglucose	NA <140	100-125 <140
NA – not applicable		
The 2- hour postglucose values are measured after a 75 gm oral glucose load		

Chronic complications of diabetes mellitus:

Many organ in the body are affected as a chronic complications of diabetes and it acts as the major cause for morbidity and mortality . There are tow types of complications - vascular and nonvascular . The vascular complications can be again subdivided into microvascular and macrovascular complications.

Non vascular complications include conditions such as gastroparesis, infections, and skin changes. Long standing diabetes may be associated with hearing loss. Among metabolic disorders of glucose diabetis mellitus is most commonly associated with auditory disfunction[12]

Damage to the nerves and vessels of the inner ear are noted in many Histopathological studies in patients with diabetes. Neuronal degeneration in the auditory system was linked to these vascular changes as an important causative factor. The organ of corti cells which are important structures of hearing are complex components and its arrangement makes it also a potential target for hyperglycaemic damage. Such damage to any part of hearing mechanism can lead to hearing loss.

Chronic complications of Diabetes Mellitus

Microvascular complications

Eye disease

- Retinopathy
- Macular edema

Neuropathy

- Sensory and motor (mono- and polyneuropathy)
- Autonomic

Nephropathy

Macrovascular complications

Coronary artery disease

Peripheral arterial disease

Cerebrovascular disease

Others

Gastrointestinal (gastroparesis, diarrhea)

Genitourinary (uropathy/ sexual dysfunction)

Dermatologic

Infections

Cataracts

Hearing loss

Glaucoma

Periodontal disease

The risk of chronic complications increases as duration and degree of hyperglycemia increases. These complications do not become apparent till the second decade of hyperglycemia. As the type 2 diabetic patients have a long asymptomatic period of hyperglycemia, they may present with complications when diagnosis is made. The microvascular changes in both type 1 and type 2 diabetes are due to chronic hyperglycemia. Clinical trials prove that reduction in chronic hyperglycemia prevents or delays retinopathy, nephropathy and neuropathy.

Evidence to prove that chronic hyperglycemia is a cause for developing macrovascular changes is less provided. However complications like coronary heart disease and mortality rates are 2 to 4 folds greater in type 2 diabetes. These complications correlate with fasting and postprandial plasma glucose levels asa well as with HbA1C values. Other factors like dyslipidemia and hypertension play an important role in macrovascular complications.

Mechanisms of complications:

Chronic hyperglycemia is a well known etiologic factor leading to complications of diabetes, the actual mechanism by which it leads to such diverse cellular and organ dysfunction is not clear. There are few prominent theories that have been enumerated to explain how hyperglycemia may lead to chronic complications of diabetes.

One theory states that raise in levels of intracellular glucose leads to the production of advanced glycosylation end products, which bind to a cell surface receptor via the nonenzymatic glycosylation of intra and extracellular proteins. The interaction of glucose with amino groups on proteins results due to nonenzymatic glycosylation . The AGEs are known to cross –link proteins, accelerate atherosclerosis, promotes glomerular dysfunction, reduces nitric oxide synthesis, induces endothelial dysfunction and leads to alteration of extracellular matrix composition and structure. Serum levels of AGEs correlates with the levels of glycemia, and these products accumulate as the glomerular filtration rate declines.

Second theory is proposed on basis of observation that hyperglycemia increases glucose metabolism by sorbitol pathway. Increased levels of sorbitol concentration alters redox potential, increases cellular osmolality leading to generation of oxygen species which is reactive and further leads to other cellular dysfunction. Trails based on this theory have lead into clinical clues on complications like retinopathy, neuropathy and nephropathy.

Third hypothesis suggests that the formation of diacylglycerol leading to activation of protein kinase C increases in hyperglycemia. This enzyme alters the transcription of genes for fibronectin, contractile protients, type IV collagen and extracellular matrix proteins.

Growth factors play an important role in diabetes related complications. Vascular endothelial growth factor (VEGF-A) is found to be increased locally in diabetic retinopathy. Other enzymes that play an important role in diabetes related complication are epidermal growth factor, platelet-derived growth factor, insulin-like growth factor, growth hormones and even insulin. Even though hyperglycemia acts as the initial trigger factor for complications of diabetes, it is still unclear if the same pathophysiologic process is effective in all organs.

Overall principles of long- term treatment:

The main goals of therapy for both type 1 and type 2 are

i) Eliminate the symptoms related to hyperglycemia
ii) Reduce or eliminate the long-term microvascular and macrovascular complications of diabetes
iii) Allow the patient to achieve a normal life as possible.

In order to achieve these goals the physician should identify a target level of glycemic control for each patient, provide the patient with educational and pharmacologic resources necessary to reach the level, and monitor or treat the diabetes related complications. Symptoms of diabetes generally resolve when the plasma glucose is < 200mg/dl.

The care of a diabetic individual requires a multidisciplinary team. Thus the term comprehensive diabetes care is used to emphasize the fact that optimal diabetes therapy involves more than plasma glucose management. Even though glycemic control is the central to optimal diabetes therapy, comprehensive diabetic care is needed to detect and manage diabetes specific complications and modify of developing diabetes associated diseases.

Treatment Regimens

A] Diet:

The fundamental element of therapy is a well-balanced, nutritious diet. The American Diabetes Association (ADA) recommendations states that about 45–65% of total daily calories should be in the form of carbohydrates; fat should be 25–35% and proteins about 10–35% . In type 2 diabetes patients the intake of carbohydrate should be limited and it should be substituted by some other calories containing monounsaturated fats, like olive oil or oils in nuts which can lower the levels of triglyceride and increase the level of HDL cholesterol. In obese individuals with type 2 diabetes, weight reduction by caloric restriction is an important goal of the diet. 300mg of cholesterol is the recommended level for both the types of diabetes.

Medications for Treating Hyperglycemia:

The medications for treating type 2 diabetes come under various categories: (1) Medications that primarily stimulate insulin secretion by attaching to the sulfonylurea receptor like Sulfonylureas, which remains as the most widely prescribed medications for treating hyperglycemia.

(2) Medications that primarily lower glucose levels by acting upon the liver, muscle, and adipose tissue like metformin which acts on the liver, thiazolidinediones which act mostly on skeletal muscles and adipose tissue. (3) Medications that principally affect absorption of glucose like α-glucosidase inhibitors acarbose and miglitol which are currently available for therapy.

(4) Medications that mimic incretin effect or prolong incretin action: Glucagon-like peptide 1 (GLP1) receptor agonists and DPP 1V inhibitors fall into this category. (5) Other: include Pramlintide that lowers glucose by suppressing glucagon and Slowing gastric emptying.

Sulfonylureas:

The main action of the sulfonylureas is to initiate the release of insulin from β- cells of pancreas. There are specific receptors on the pancreatic β- cells surface which bind sulfonylureas in the order of insulinotropic potencity. It has been observed these receptors closes the potassium channels leading to depolarization of the β cell when they are activated. This depolarized state allows calcium to enter into the cell and this promotes the release of insulin.

Sulfonylureas are not recommended for use in type 1 diabetic individuals as they require functioning β cells in pancreas to deliver their effect on blood glucose. These medications are used in type 2 diabetes patients who show improvement during acute administration. This results in improvement in early phase of release of insulin which serves as refractory agent in acute glucose stimulation. It is metabolized in the liver.

The metabolites are excreted by the kidney and, in the case of the secondgeneration sulfonylureas, partly excreted in the bile. Sulfonylureas are generally contraindicated in patients with severe liver or kidney impairment.

Metformin:

This is used alone or along with other oral hypoglycemic agents or insulin, in the treatment of type 2 diabetes patients. Metformin activates adenosine monophosphate-activated protein kinase (AMPK) by uncoupling mitochondrial oxidative phosphorylation and increasing cellular AMP levels. Metformin’s therapeutic effects primarily derive from the increasing hepatic AMPK activity, which reduces hepatic gluconeogenesis and lipogenesis. Metformin is a substrate for organic cation transporter 1, which is abundantly expressed in hepatocytes and in the gut. 1.5–3 hours is the half life of metformin. It does not bind to plasma proteins. It is excreted unchanged by the kidneys as it cannot be metabolized in humans. Metformin is the first-line therapy for patients with type 2 diabetes.

The current recommendation is to start this medication at diagnosis. An added benefit of metformin treatment is that has propensity to maintain both fasting and postprandial hyperglycemia and hypertriglyceridemia in obese patients with diabetics without the complication of weight gain which is a known complication in treatment with insulin or sulfonylurea. Metformin is not effective in patients type 1 diabetes patients. Patients with chronic kidney disease should not be given this medication because failure to excrete it would produce high blood and tissue levels of metformin that could stimulate lactic acid overproduction. Also patients with liver failure or persons who abuse alcohol should not receive this medication because it could result in lactic acidosis.

500 mg, 850 mg, and 1000 mg tablets are the available doses of metformin. 500 mg and 750 mg doses are available as extended-release formulations. Even though the maximum dose is 2.55 gm, very less benefit is observed in doses more than 2000 mg. It is necessary to start with lower doses and then to increase the dosage very slowly in divided doses which is taken with meals so as to reduce the minor complications like gastrointestinal upsets.

The frequent side effects of metformin are gastrointestinal symptoms like anorexia, nausea, vomiting, abdominal discomfort, diarrhea which occur in up to 20% of patients. These effects are dose related, tend to occur at onset of therapy, and often are transient side effects. Hypoglycemia does not manifest with treatment of metformin, so it permits to maintain an "euglycemic" state. So it is termed as "antihyperglycemic" medication.

Certain individuals receiving radiocontrast agents may manifest with acute kidney failure as a rare complication. Therefore it is recommended to with hold therapy with Metformin temporarily on the day of radiocontrast administration and it should be restarted a day or two later after confirmation that renal function has not deteriorated.

Thiazolidinediones:

Medications in this class of antihyperglycemic agents sensitize insulin in peripheral tissues. They bind a nuclear receptor called peroxisome proliferator-activated receptor gamma (PPAR-) and affect the expression of a number of genes. Observed effects of thiazolidinediones include increased glucose transporter expression, decreased free fatty acid levels, decreased hepatic glucose output, increased adiponectin and decreased release of resistin from adipocytes, and increased differentiation of preadipocytes into adipocytes.

It is also proven to reduce the levels of plasminogen activator inhibitor type 1, C-reactive protein matrix metalloproteinase 9 and interleukin 6. This class of medications does not cause hypoglycemia. The two drugs available for therapeutic use are Rosiglitazone and pioglitazone. They can be as monotherapy as well as in combination with sulfonylureas; metformin or insulin, for lowering levels of HbA1c. In instances where a combination is used, the dosage of insulin can be reduced by 30-50% and slowly insulin can be stopped.

The dosage of rosiglitazone is 4–8 mg daily and of pioglitazone, 15–45 mg daily, and the medication need not be taken with food. When thiazolidinedione and metformin are togeather the advantage hypoglycemia can be avoided. Patients who do not get adequate blood sugar control with sulfonylureas, generally maintain well with combination

Insulin

Insulin is indicated for type 1 diabetes as well as for type 2 diabetic patients with insulinopenia whose hyperglycemia do not respond to diet therapy alone or combined with other hypoglycemic medications. With the development of highly purified human insulin preparations, immunogenicity has been reduced markedly, thereby decreasing the incidence of therapeutic complications such as insulin allergy, immune insulin resistance, and localized lipoatrophy at the injection site.

It has been impossible to replicate the patterns of secretion of intraportal insulin with short-acting or longer-acting insulin preparations given as subcutaneous injections. Even if correction in diet and exercise are done routinely, along with capillary blood glucose level monitoring at home, control in blood glucose levels is possible only on using many mixtures of insulin at least two times a daily or a portable infusion pumps should be used.

Characteristics of available insulin preparations:

Commercially available preparations of insulin vary in terms of onset of time their biologic duration of action:

A. Species of insulin: Recombinant DNA techniques are used to produce human insulin, which is widely available in the trade name of Novolin. It is marketed as either regular or NPH formulations.

Human insulin are available in five analogs, out of which three are rapidly acting, example insulin lispro, insulin glulisine and insulin aspart and two are long-acting example insulin glargine and insulin detemir. The FDA has given approval for clinical use of these drugs. Insulin derived from animal source is not used anymore in the United States.

B. Purity of insulin: "Purified" insulin is the one which has proinsulin contamination < 10 ppm and is the degree of purity. The same is recommended by FDA. Now a day's all available insulin forms contain < 10 ppm of proinsulin and are termed as "purified". These forms of insulins preserve their potency well, making refrigeration not very crucial. This property makes it easy to carry reserve supplies of insulin during traveling without losing its potency, but it has to be protected from direct heat or cold.

C. Concentration of insulin: Currently in the United States, the available concentrations of insulin are 100 units/mL and it is dispensed as 10-ml vials. The low-dose (0.5- or 0.3-mL) disposable insulin syringes are now very popular. Doses as low as, 1–2 units can be measured accurately.

U500 regular human insulin (Humulin R) is available in rare insistences where there is severe insulin resistance

Methods of insulin administration:

A. **Insulin syringes and needles**: Disposable plastic syringes in sizes 1 ml, 0.5 ml, and 0.3 ml are available. The 0.3 ml low dose syringes have gained popularity now a day's as majority of diabetic patients take less than 30 units of insulin as a single injection except in conditions like extreme insulin resistance which is a rare entity. Needles are available in 2 lengths, short are 8 mm and long is 12.7 mm. Long needles are used in patients who are obese to decrease the variability in absorption of insulin. These needles are ultrafine and size is small about 31 gauges, so pain of injection is much reduced. Disposable syringes can be used upto three to five times till the blunting of the needle is noted. Sterility can be maintained by recapping syringes between uses, to avoid infection with reuse.

Needle cleansing with alcohol is avoided as it can dissolve the coating of silicon which may increase the pain while puncturing the skin. Any body part which is covered by loose skin, like abdomen, upper arms, flanks, thighs, and upper buttocks can be chosen. Skin preparation with alcohol is no longer recommended required before injection as far as the skin is kept clean continues to be recommended To avoid delayed absorption rotation of sites is recommended as it causes fibrosis or lipohypertrophy due to repeated injections. However variability in absorption rates are noted in different sites. Subcutaneous injections are preferred to be given in the abdomen as regular insulin is found to absorb more rapidly. In analog type insulin preparations the effect of anatomic regions appear to be much less

A. **Insulin pen injector devices**: The need for carrying insulin vials and syringes are eliminated since the advent of insulin pens. Cartridges of insulin lispro, insulin aspart, and insulin glargine are available as reusable pens. Disposable prefilled pens are also available for insulin lispro, insulin aspart, insulin glulisine, insulin detemir, insulin glargine. Thirty-one gauge needles (5, 6, and 8 mm long) for these pens make injections almost painless.

B. **Insulin pumps:** Medtronic Mini- Med, Animas, Insulet, and Roche are some of the companies that make insulin infusion pumps for subcutaneous delivery of insulin. These pumps are small and are very easy to program. They offer many features, including the ability to set a number of different basal rates throughout the 24 hours and to adjust the time over which bolus doses are given. They also detect pressure build-up if the catheter is kinked. Improvements have also been made in the infusion sets. The catheter connecting the insulin reservoir to the subcutaneous cannula can be disconnected, allowing the patient to remove the pump temporarily. Ominpod is an insulin infusion system in which the insulin reservoir and infusion set are integrated into one unit so there is no catheter. The pod, placed on the skin, delivers subcutaneous basal and bolus insulin based on wirelessly transmitted instructions from a personal digital assistant. The advantage of continuous subcutaneous insulin infusion (CSII) is that it allows establishment of a basal profile tailored to the patient. The patient therefore is able to eat with less regard to timing because the basal insulin infusion should maintain constant blood glucose between meals. Also the ability to adjust the basal insulin infusion makes it easier for the patient to manage glycemic excursions that occur with exercise.

The pumps also have software that can assist the patient to calculate boluses based on glucose reading and carbohydrates to be consumed. They can keep track of the time elapsed since last insulin bolus and the patient is reminded of this when he or she attempts to give additional correction bolus before the effect of the previous bolus has worn off. This feature reduces the risk of overcorrecting and subsequent hypoglycemia.

RATIONALE FOR STUDY

Our institution, PSG Institute of Medical Science & Research in Coimbatore serves as a referral centre for the southern states of India like Tamil Nadu, Kerala and Andhra Pradesh which have an agricultural economy. The modern lifestyle modification has led to many problems like obesity, early onset diabetes, dyslipidemia, coronary artery disease. Among these diabetes mellitus is found to be on the rise. This multi-system abnormality causes many irreversible changes in the body leading to many complications including hearing loss which is given less importance or neglected in many situations.

Reviewing the available literature we realized that all important studies on prevalence of hearing loss among diabetic patients were done in the Europe and American Continents. But the incidence of diabetes mellitus and its complications in India has been steadily increasing. There is a lack of recognition of hearing loss as a complication of diabetes. It is this lack of awareness and need for screening of these individuals at higher risk of developing hearing loss, that prompted us to undertake this study.

MATERIALS & METHODS

This is a cross-sectional study in which evaluation of 200 patients who were diagnosed with diabetes mellitus undergoing treatment on Out–Patient or In-Patient basis in department of E.N.T and Endocrinology, in PSG Institute of Medical Science & Research, Coimbatore, Tamil Nadu from July 2012 to July2014 were considered.

History of every patient was taken in detail and importance was given on their presenting complaints. Any significant past history of ear discharge, hearing loss or previous ear surgeries were elicited. History of duration of diabetes and mode of treatment were given importance.

All the patients were taken up for Otoscopic examination, in which the ear canal and status of tympanic membrane were assessed.

Inclusion Criteria for cases considered in the study:

1. Known cases of Diabetes mellitus
2. Age-group >30 years to <60 years
3. Both genders

Exclusion Criteria for cases:

1. Individuals involved in occupations exposing to loud noise
2. Individuals with previous history of ear discharge/hearing loss/tinnitus/ear surgery
3. Otoscopic examination showing any ear pathology like wax, discharge, retracted tympanic membrane, perforated tympanic membrane or tympanosclerosis.
4. Patients unwilling to comply with the study.

Evaluation:

All 200 patients were worked up with a Pure tone audiomertry. The audiogram was analyzed for hearing loss and if present it will be classified according to WHO classification.

Pure tone audiometery:

Pure Tone Audiometry is an audiometric evaluation which is done routinely. The results of which are charted in the form of audiogram which is used to describe the degree of hearing loss. In our study audiological examination was carried out in a sound proof room using the Pure Tone Audiometer model GSI 61 in the ENT department, PSG Institute of Medical sciences & research. To test the air conduction ear phones were used and a small vibrator, was used to test hearing by bone conduction which was placed over the mastoids. The signals presented to the subject by the audiometer were all characterized by The sound pressure level, frequency and wave form were all delivered at controlled rates to the subjects which was presented in the form of signals by the audiometer.

The entire purpose and procedure of the test was explained in detail to the patients. On daily basis before starting the procedure biological calibration was done for both air and bone conduction.

Pure Tone Audiometery: Air Conduction Threshold

Here a range of specific puretones are delivered to the patient through earphones and threshold for hearing is measured. Hughson – Westlake method is followed which is an ascending order.

up 5, down 10 method is done

Test frequencies

An electronic device that delivers pure tones is called as an audiometer. The intensity of these tones can be either increased or decreased in steps of 5-db. The thresholds that are measured for air conduction are 250, 500, 1000, 2000, 4000 6000 and 8000 Hertz. The thresholds for bone conduction are measured for 250, 500, 1000, 2000, 4000 Hz. The patient was briefed that tones of short duration would be heard in either the left or the right ear and these sounds to begin with are very faint. It was also explained that response for these sounds should be in the form of raising his finger incorrespondance to the side of the ear, as in which the tone was heard. The hand should be kept raised as long as it is heard, even if faint tone was heard.

Threshold determination:

The test is started at 1000 Hz. A clearly audible signal, about 40 dB HL will be presented to the individual assuming that the hearing threshold of the subject is within normal limits. If the patient finds it difficult to hear then it is raised to 60 dB HL. The tone levels will then be reduced in 10 dB steps till the tone became inaudible and the patient is not responsive. Next the level of tones will be raised in 5 dB steps, delivering one pulse in each level until obtaining a response. The point at which the individual makes a response on raising it by 5 dB will be considered as the threshold. The same test will be done at the higher frequency till 8000 Hz. Again the threshold is brought back to 1000 Hz and then further lowered to 500 Hz and 250Hz to be tested. Then the opposite ear will be tested in a similar fashion.

Pure Tone Audiometry: Bone Conduction Threshold Bone conduction in pure tone audiometry is performed in combination with air conduction audiometry. This provides information about the elements of conduction in hearing loss. In this test hearing thresholds are measured for pure tones which are delivered with the help of a bone vibrator placed over the mastoid process behind the ear. The measurement was done by Hughson Westlake ascending up 5, down 10 method.

Procedure:

Thresholds like 250, 500, 1000, 2000, 4000Hz are measured for bone conduction. The patient was briefed that he would hear short tones that may be very faint and it might be simultaneously heard in any one ear or both ears . The response to this is done by lifting the finger corresponding ear in which he hears, as soon as he perceives the tone. He was also briefed about the bone vibrator placed over the mastoid which not supposed to be manipulated after it is placed finally.

Threshold determination:

In this test, the better ear is tested first, once the side difference is found out by prior testing or by Weber's test to determine the side of lateralization. The mastoid process is selected for the placement of bone vibrator. Initially test is done at 1000 Hz. An audible tone which is clear and continuous is delivered and continous adjustments on the bone vibrator are done till the patient denotes that it is the loudest tone. It was made sure to avoid contact between the vibrator and the outer ear. In this test earphones are not provided. At about 40 dB test tones will be delivered for 1to 2 seconds and if it is at inaudible levels then, test tone will be increased in 10 dB steps until a response was noted. This is again repeated but by reduceing the levels 20 dB steps, until it becomes inaudible and no response was obtained from the patient. This is followed by raising levels in 5 dB steps, till the patient responds. The threshold level at which the patient responds is taken into consideration. The other test frequencies are also test as the test continues. The other ear is also tested. Masked pure tone audiometry has to be done if the difference of more than 40 dB between air conduction threshold of the test ear and the bone conduction threshold of the opposite ear or when the air bone gap of the poorer ear is more than 10 dB.

RESULTS

In this study, analysis of 200 patients diagnosed with diabetes mellitus was done in the Department of E.N.T, PSG Institute of Medical Sciences & Research from July 2012 to July 2014 to determine the incidence of hearing loss as a complication of diabetes mellitus. Detailed evaluation of each case was done comprising of the history, clinical examination including otoscopic examination with otoscope and pure tone audiometry.

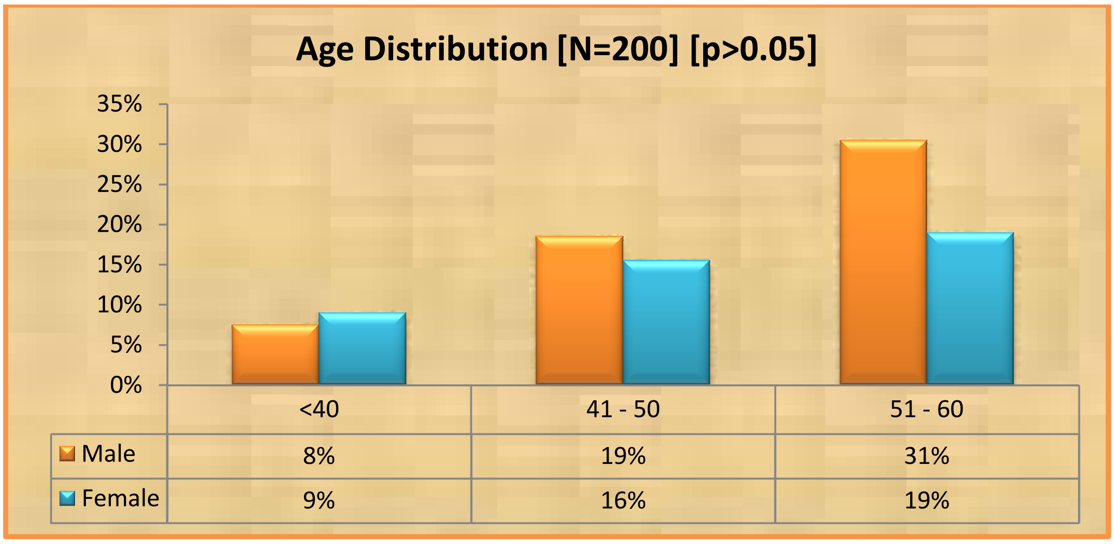
The clinical data was collected by means of a proforma and the observations from the audiogram were analyzed with the Master chart as shown in the Annexure.

The results have been evaluated primarily keeping in mind the aim of the study, to determin the prevalence of Hearing loss and its association with Diabetes and to assess the hearing loss in diabetics and correlate it with age and duration of diabetes

In our study of 200 diabetic patients, 110 (55%) cases turned out to have sensorineural hearing loss.

Age distribution of Diabetes Mellitus cases:

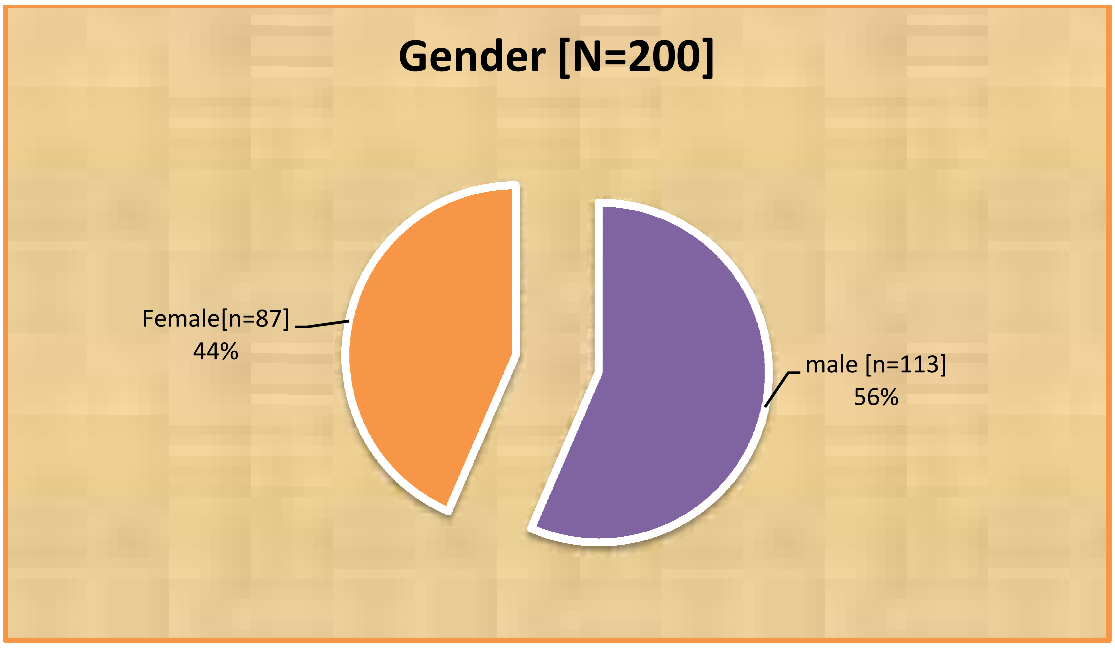
Age Distribution			
	Gender		Total
Age	Male	Female	
<40	15	18	33
41 - 50	37	31	68
51 - 60	61	38	99
Total	113	87	200



From the above mentioned table it is evident that the maximum incidence of patients with diabetes (50%) was in the group 51-60 years, followed by 35% in 41-50 years.

Sex distribution of Diabetes Mellitus cases:

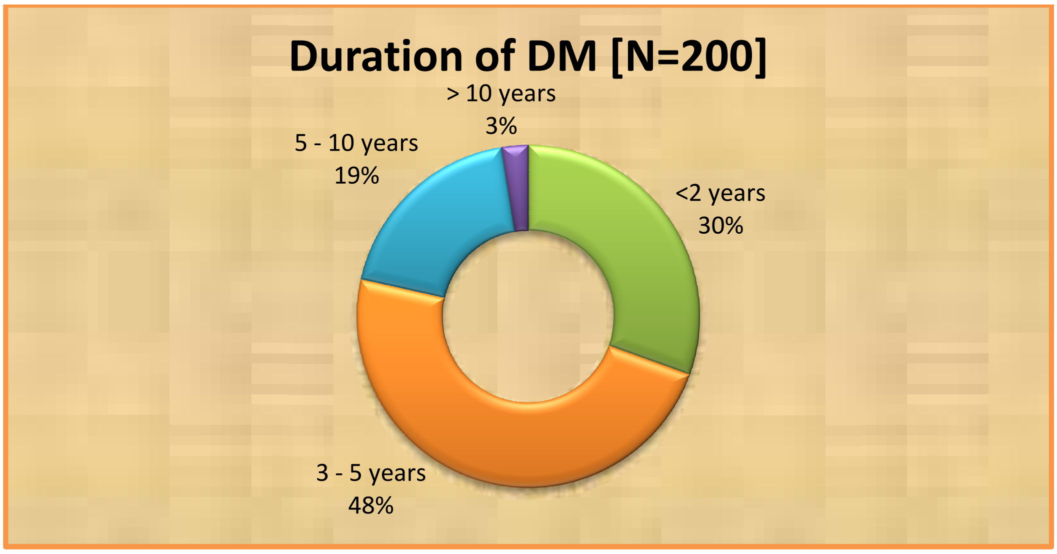
Sex	No. of cases	Percentage (%)
Male	113	56
Female	87	44
Total	200	100



In our study, the incidence of patients with diabetes mellitus amongst males was 56% (113 males) and 44% in females (87 females)

Overall duration of Diabetes Mellitus in our study:

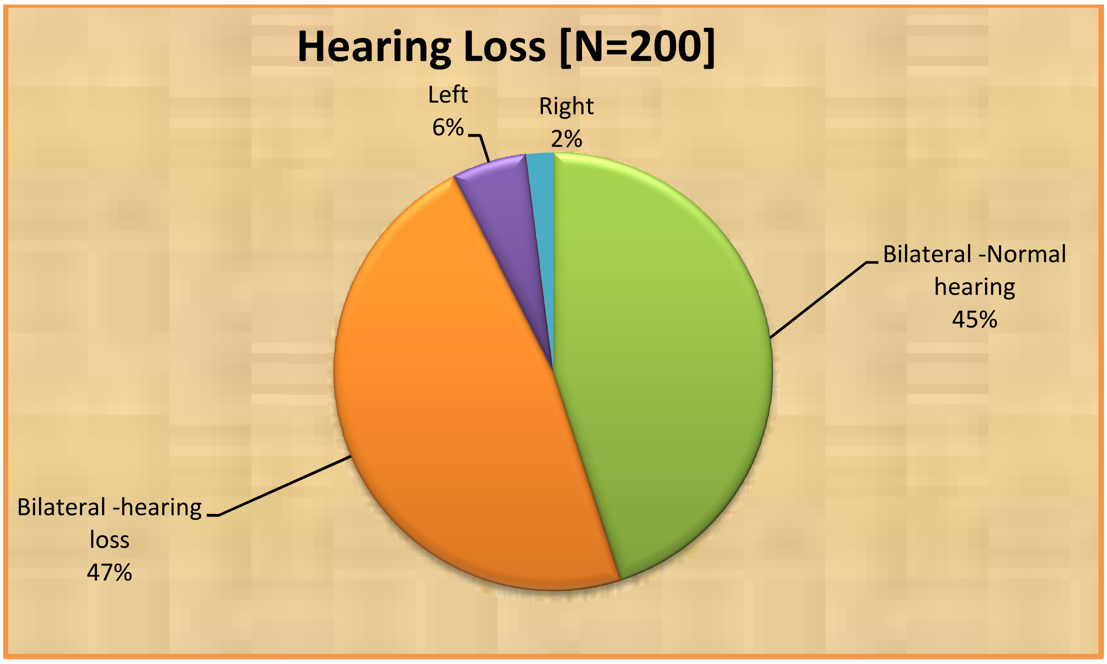
Duration of DM		
Duration of DM	n	(%)
<2 years	61	31%
3 - 5 years	96	48%
5 - 10 years	38	19%
> 10 years	5	3%
Total	200	100%



In our study, the incidence of diabetes mellitus is most between 3-5 years 48%, followed by less than 2 years 30%; 19% for 5-10 years and only 3% for less than 10 years.

Overall distribution of hearing in our study:

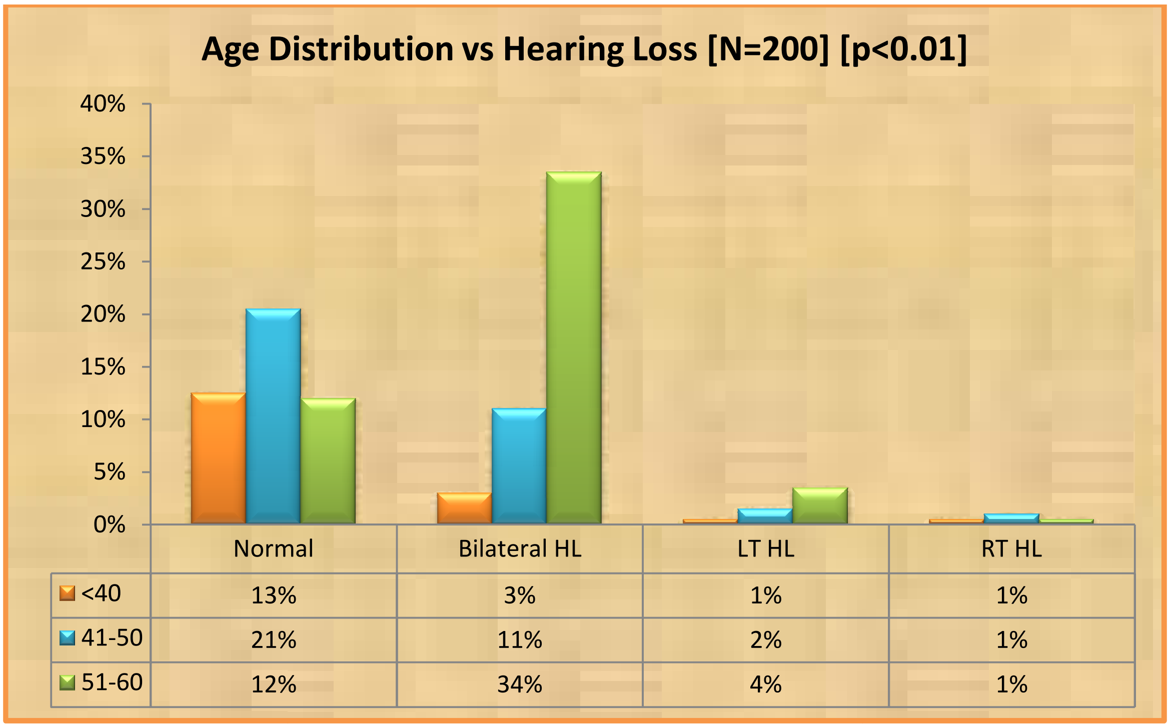
`Hearing Loss		
Hearing Loss	n	(%)
Bilateral -Normal hearing	90	45%
Bilateral -hearing loss	95	47%
Unilateral hearing loss		
Left	11	6%
Right	4	2%
Total	200	100%



In our study out of 200 diabetic patients,110 patients (55%) had sensorineural hearing loss, out of which 95 patients (47%) had bilateral sensorineural loss and 15 patients (8%) had unilateral hearing loss, in which 11 (6%) had in the left ear and 4 (2%) on the right ear. And 90 patients (45%) had hearing within normal limits

Overall distribution of hearing loss with age:

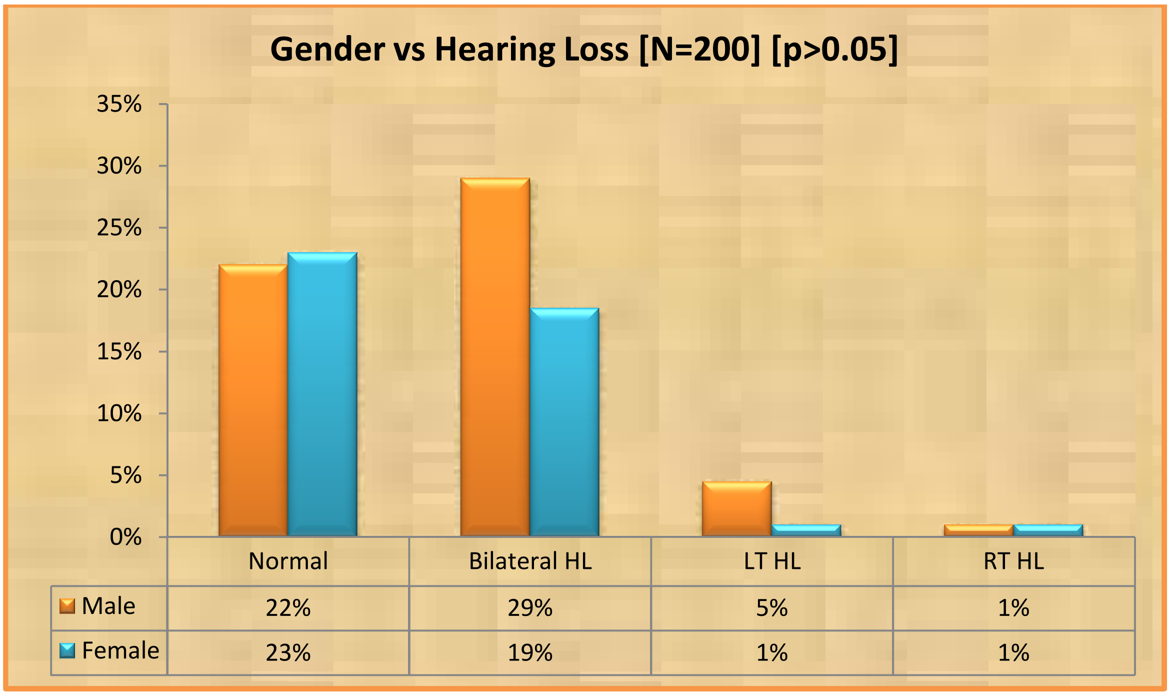
Hearing Loss with Age Distribution					
	Out come				
age group	Normal	Bilateral HL	LT HL	RT HL	Total
<40	25	6	1	1	33
41-50	41	22	3	2	68
51-60	24	67	7	1	99
Total	**90**	**95**	**11**	**4**	**200**



In our study, maximum number of diabetic patients with hearing loss was noted among 51-60 years which was 99 cases, followed by 41-50 years (68 cases) and only 33 cases among less than 40 years of age.

Distribution of hearing loss with gender:

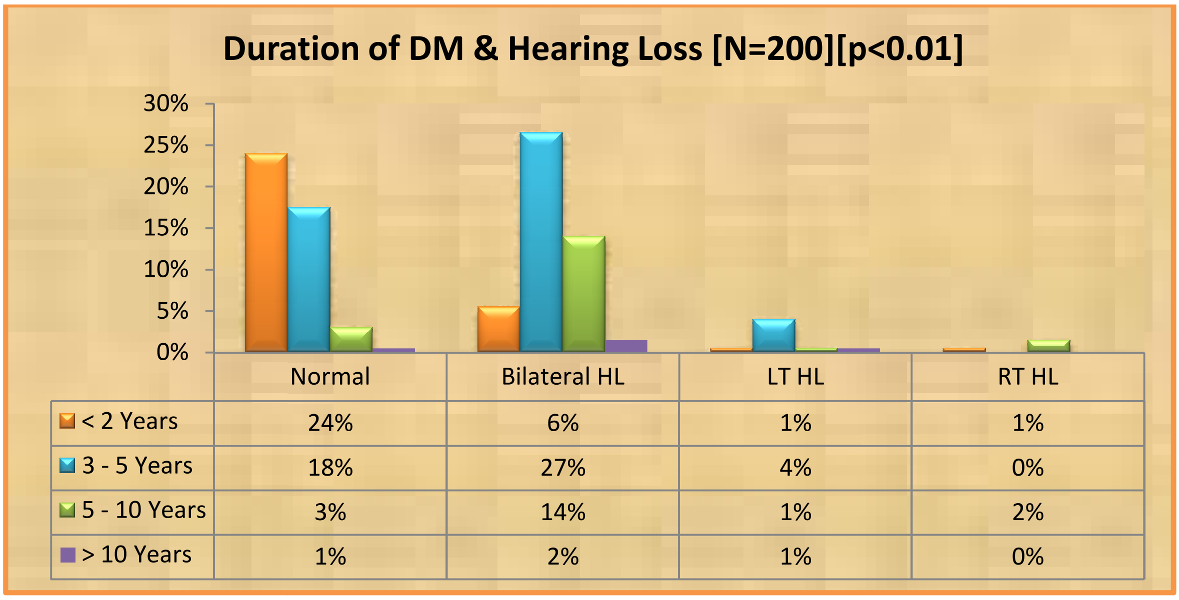
Hearing Loss with Gender					
	Out come				
Gender	**Normal**	**Bilateral HL**	**LT HL**	**RT HL**	**Total**
Male	44	58	9	2	113
Female	46	37	2	2	87
Total	**90**	**95**	**11**	**4**	**200**



In our study, it was noted that more number of males 113(56%) had hearing loss comparing to that of females who were only 87 (44%). The p-value was >0.05, which makes it statically not significant.

Distribution of hearing loss with duration of Diabetes Mellitus:

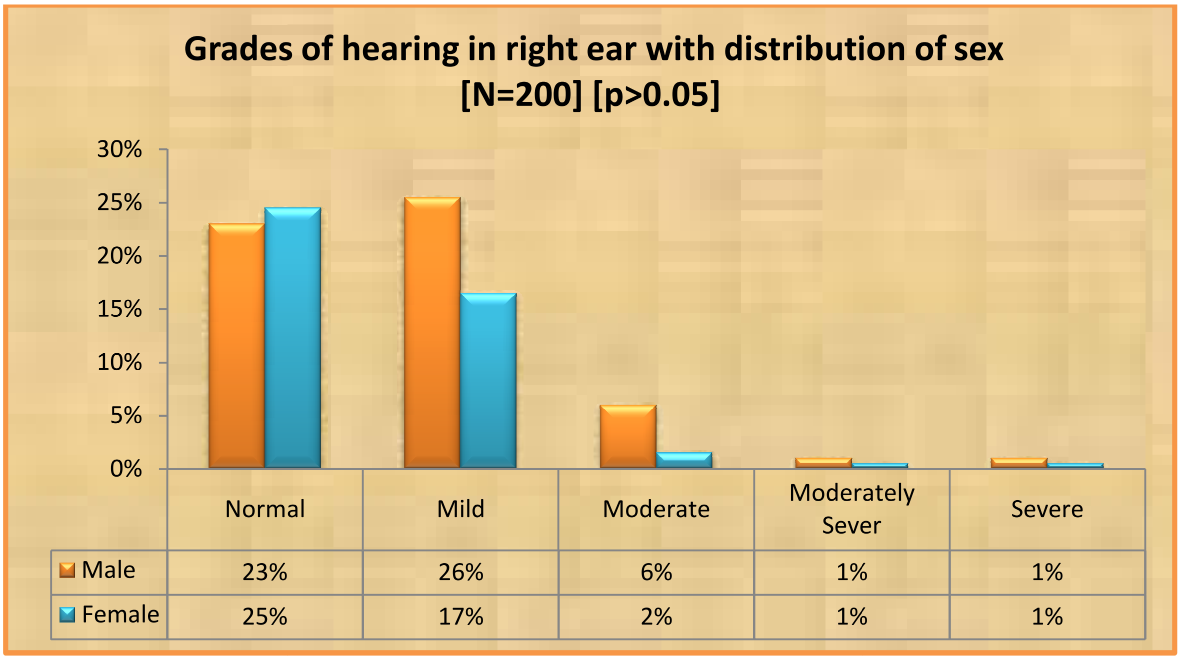
Hearing Loss with DM Duration					
	Out come				
DM Duration	**Normal**	**Bilateral HL**	**LT HL**	**RT HL**	**Total**
< 2 Years	48	11	1	1	61
3 - 5 Years	35	53	8	0	96
5 - 10 Years	6	28	1	3	38
> 10 Years	1	3	1	0	5
Total	**90**	**95**	**11**	**4**	**200**



From the above mentioned table, it is noted that the incidence of hearing loss was found to be more among patients with diabetes for 3-5 years, which was 96, followed by 38 cases between 5-10 years and only 5 cases with diabetes for more than 10 years. The p-value is <0.01, which is statically significant.

Grades of hearing loss in right ear with sex distribution:

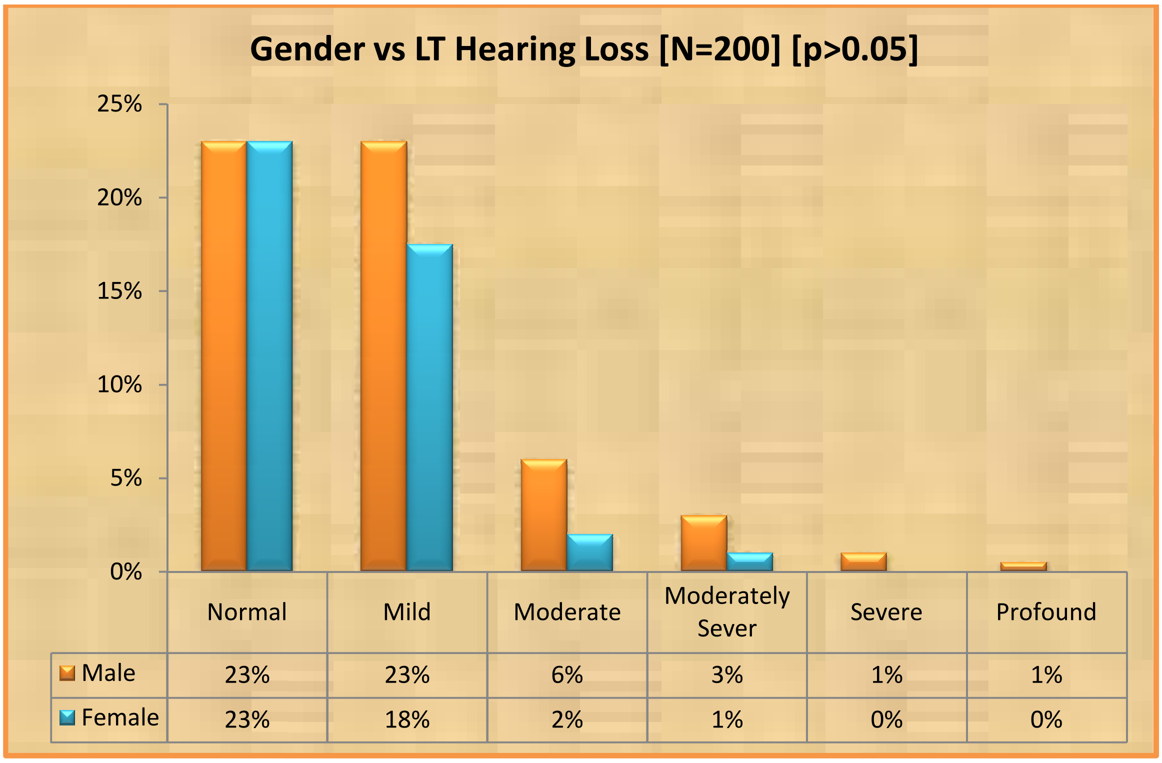
Right Side Ear - Hearing Loss						
Gender	**Normal**	**Mild**	**Moderate**	**Moderately Severe**	**Severe**	**Total**
Male	46	51	12	2	2	113
Female	49	33	3	1	1	87
Total	**95**	**84**	**15**	**3**	**3**	**200**



It is noted that in the right ear 95 cases had normal hearing, 84 cases had mild hearing loss, 15 had moderate and 3 cases had moderately severe and severe hearing loss respectively.

Grades of hearing loss in left ear with sex distribution:

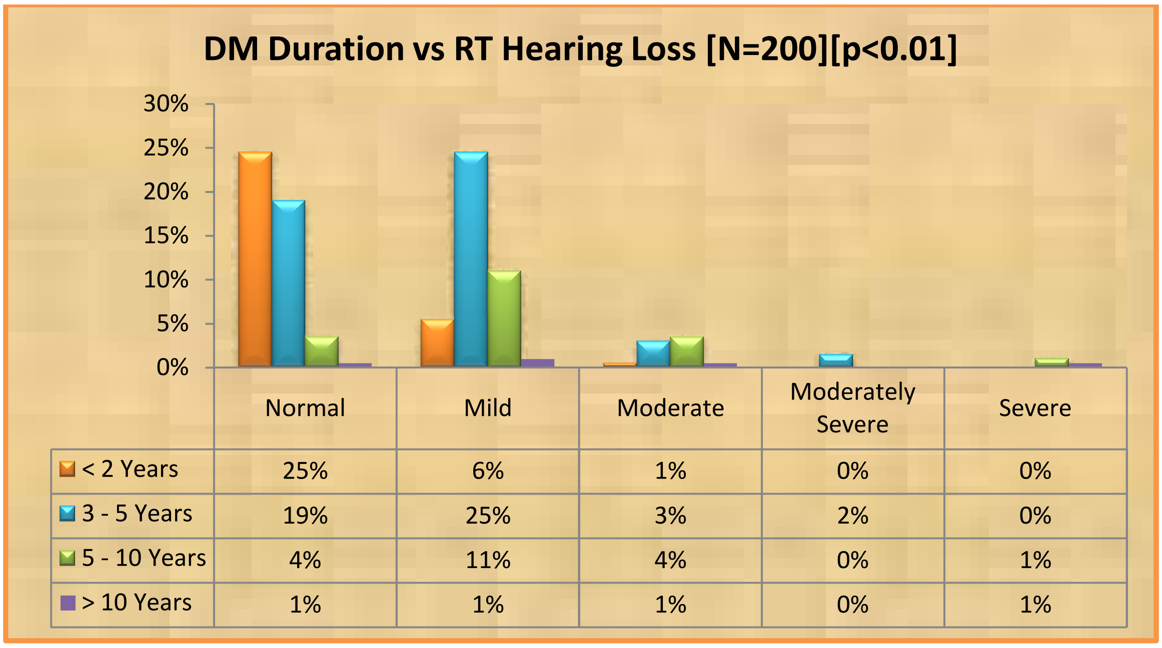
	Left Side Ear - Hearing Loss						
Gender	**Normal**	**Mild**	**Moderate**	**Moderately Severe**	**Severe**	**Profound**	**Total**
Male	46	46	12	6	2	1	113
Female	46	35	4	2	0	0	87
Total	**92**	**81**	**16**	**8**	**2**	**1**	**200**



It is noted that in the left ear, 92 cases had normal hearing, 81 had minimal hearing loss, 16 had moderate loss 8 moderately severe, 2 with severe and 1 with profound hearing loss. Thus statically not significant ($p>0.05$)

Severity of hearing loss in right ear with duration of diabetes mellitus:

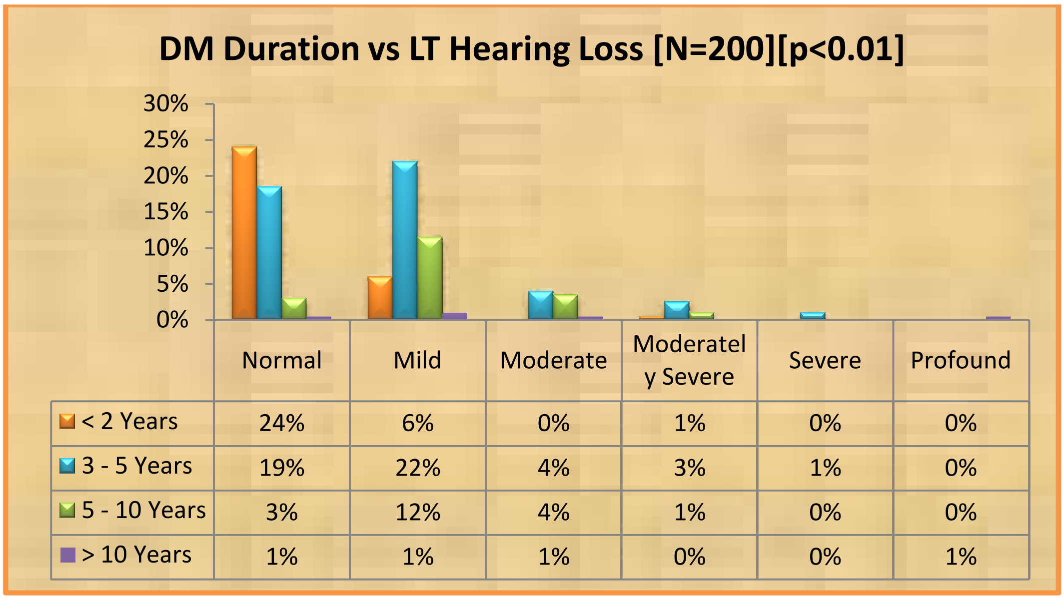
Right Side Ear - Hearing Loss						
DM Duration	**Normal**	**Mild**	**Moderate**	**Moderately Severe**	**Severe**	**Total**
< 2 Years	49	11	1	0	0	61
3 - 5 Years	38	49	6	3	0	96
5 - 10 Years	7	22	7	0	2	38
> 10 Years	1	2	1	0	1	5
Total	**95**	**84**	**15**	**3**	**3**	**200**



From the above mentioned table, it is noted that maximum of 58 (30%) cases had hearing loss among the group 3-5 years, among which 49 (25%) cases had mild hearing loss; 6 (3%) cases had moderate hearing loss and 3 cases (2%) had moderately severe loss, followed by 5-10 years group where 31 cases (16%) had hearing loss.

Severity of hearing loss in left ear with duration of diabetes mellitus:

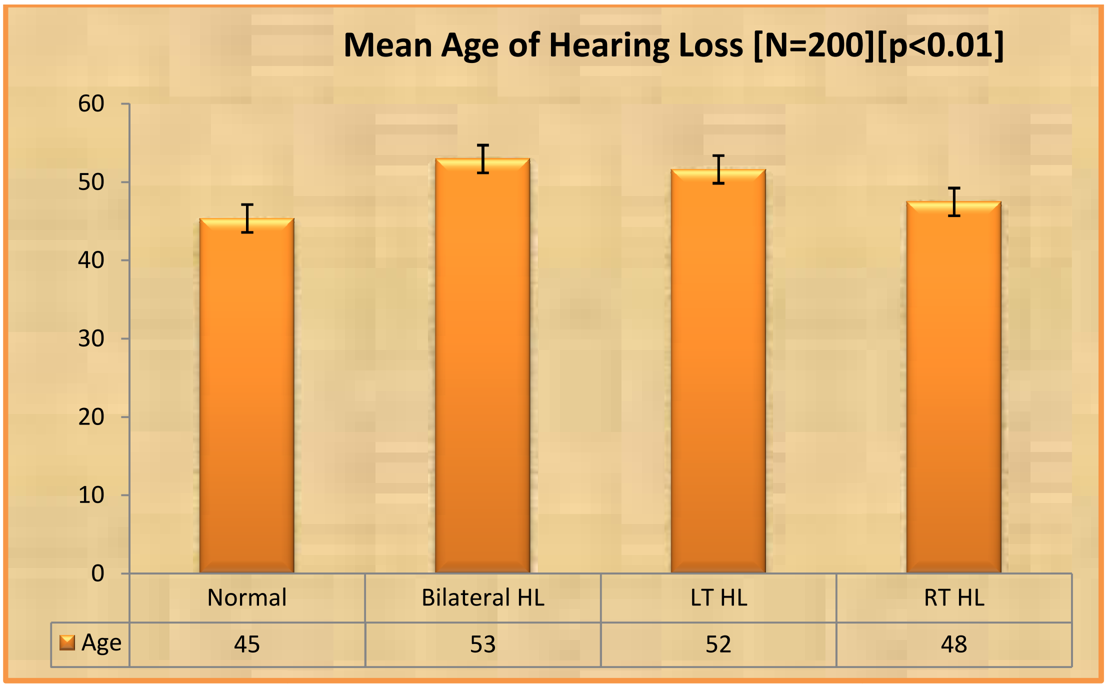
	Left Side Ear - Hearing Loss						
DM Duration	**Normal**	**Mild**	**Moderate**	**Moderately Severe**	**Severe**	**Profound**	**Total**
< 2 Years	48	12	0	1	0	0	61
3 - 5 Years	37	44	8	5	2	0	96
5 - 10 Years	6	23	7	2	0	0	38
> 10 Years	1	2	1	0	0	1	5
Total	**92**	**81**	**16**	**8**	**2**	**1**	**200**



From the above mentioned table, it is noted that, maximum of 59 (30%) cases had hearing loss among the group 3-5 years, among which 44 (22%) cases had mild hearing loss; 8 (4%) cases had moderate hearing loss and 5 cases (3%) had moderately severe loss, followed by 5-10 years group where 32 cases (17%) had hearing loss.

Mean age of hearing loss:

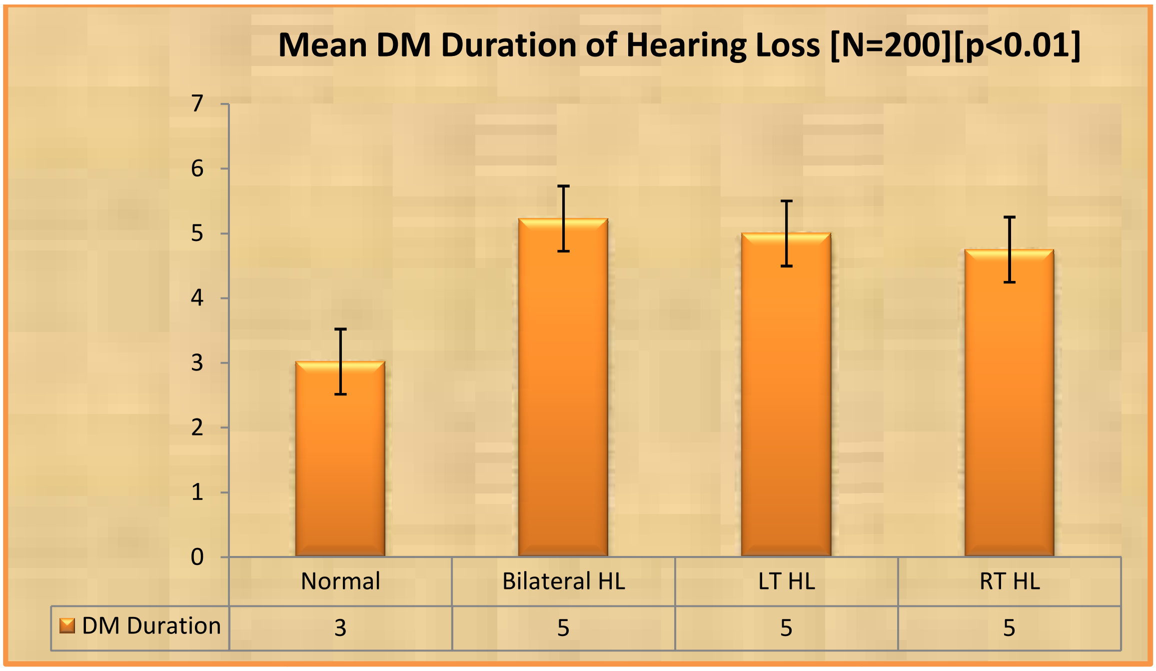
Mean Age of Hearing Loss							
Hearing Loss	N	Mean	SD	95% CI for Mean Lower Bound	95% CI for Mean Upper Bound	Minimum	Maximum
Normal	90	45	8	44	47	30	60
Bilateral SNHL	95	53	6	52	54	36	60
LT HL	11	52	7	47	57	34	60
RT HL	4	48	8	35	60	39	58
Total	200	49	8	48	50	30	60



In our study it is noted that maximum incidence of hearing loss was noted among age group 50 to 55 years of age. The p-value is <0.01, thus making it statically significant.

The mean distribution of hearing loss with duration of diabetes:

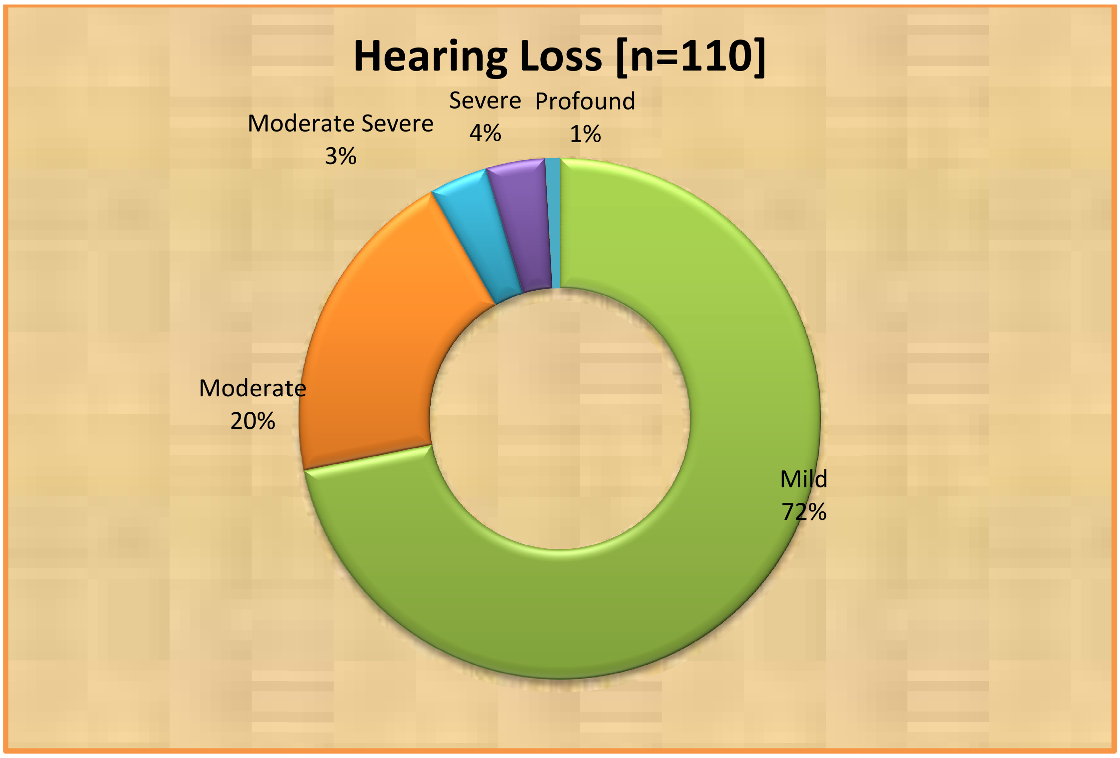
Mean DM Duration of Hearing Loss							
	N	Mean	SD	95% CI for Mean		Minimum	Maximum
				Lower Bound	Upper Bound		
Normal	90	3	3	2	4	1	20
Bilateral SNHL	95	5	3	5	6	1	25
LT HL	11	5	4	3	7	2	15
RT HL	4	5	3	1	9	1	6
Total	200	4	3	4	5	1	25



In our study, it is noted that maximum incidence of hearing loss is noted among diabetic patients with 3- 5 years duration. The p- value is <0.01, thus making it statistically significant.

Overall severity of hearing loss in our study:

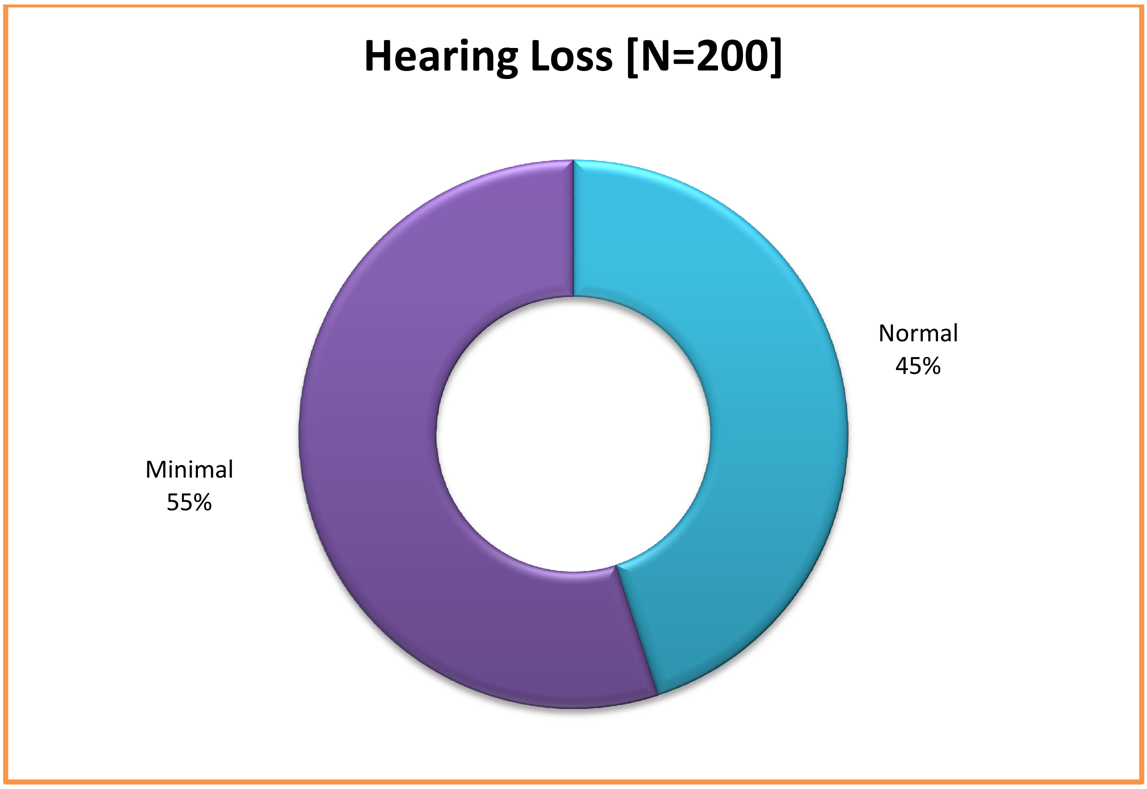
Hearing Loss [n=110]			
Hearing Loss	n	(%)	
Mild	79	72%	
Moderate	22	20%	
Moderate Severe	4	4%	
Severe	4	4%	
Profound	1	1%	
Total	110	100%	



In our study, it is noted that 72% of individuals with diabetes have mild sensorineural hearing loss, followed by 20% with moderate sensorineural hearing loss.

Overall incidence of hearing in our study:

	Hearing Loss		
	Normal	Hearing loss	
No. of patients	90	110	
Percentage	45%	55%	



From the above mentioned table, it is shown that, in our study 55% of diabetic patients were found to have sensorineural hearing loss and 45% of cases had hearing within normal limits.

DISCUSSION

Hearing loss is a dysfunction of hearing and it varies in severity ranging from mild; moderate; severe or profound. Hearing loss in general can be divided into conductive type, sensorineural type or mixed type. The hearing impairment typically seen among diabetic patients is sensorineural type and bilateral. Diabetes mellitus has been proven an individual causative factor in developing sensorineural type of hearing loss [14].

Diabetic patients are found to have different types of hearing loss. One among them is gradually progressive and bilateral sensorineural type, mainly effecting high frequencies in elderly patients. This may be as same as presbyacusis, but with a loss more severe than expected because of aging [15].

Acquired hearing loss is defined as a loss of hearing function due to some nongenetic causes, for example triggered by environmental agents like chemicals, drugs and noise. Presbycusis or age- related hearing loss may have both a genetic and an acquired component.

Sensorineural hearing loss results from lesions of cochlea, VIIIth nerve or central auditory pathway. Characteristically these patients have poor speech discrimination and difficulty in hearing in the presence of noise. The pathway for sound impulses from hair cells to inner ear to the auditory nerve and the brain is damaged It may be acute (sudden) or chronic sensorineural hearing loss.

Common causes of acquired sensorineural hearing loss are:

- Age related hearing loss
- Acoustic trauma
- Viral infections of the inner ear
- Meniere's disease
- Drugs like aspirin, quinine, and aminoglycoside antibiotics, which can affect the hair cells
- Neural deafness may be due to multiple sclerosis, brain tumor and stroke.
- Systemic causes like diabetes, hypothyroidism, artherosclerosis

AGE:

Our study has studied the pattern of hearing loss in diabetic patients and their effect of hyperglycemia on hearing thresholds and it shows 55% incidence of deafness. And the peak age group being affected with hearing loss in our study was between 50- 53 years of age.

In contrast a study conducted by Rajendran S , Anandhalakshmi et al[16] showed maximum incidence between 40- 50 years of age. On further review, we realize that acquired on set sensorineeural hearing loss can occur early in diabetic patients. Usually age related sensorineural deafness is more common in people more than 60 years of age. But here it is seen that diabetic patients are at higher risk of developing this sensory loss at a much younger age groups.

It is also noted that in our study there were more people with diabetes among the age group 51-60 years of age (50%).The p value is <0.05, which makes it statistically significant.

Sex:

In our study it was noted that more number of diabetic males were affected with hearing loss, than females. In contrast Taylor and Irwin (1978) noted that diabetics who are females had a significantly higher hearing loss on comparing with diabetic male patients.

But according to Cullen and Cinnamond (1993) [17] diabetic male patients had hearing worse than diabetic female patients

From our study, we conclude that sex of an individual is not statistically significant ($p>0.05$) parameter in considering the susceptibility to develop hearing loss.

It was also noted that out study of 200 diabetic patients, 113(56%) were males and 87(44%) were females. This shows that males are more prone to develop Diabetes mellitus.

Overall incidence of hearing loss in diabetics:

In our study, we evaluated the hearing loss in diabetic patients and their effect of hyperglycemia on hearing loss and it shows a 55% incidence of deafness. Friedman et al [18] observed an incidence of 55% hearing loss in patients with diabetes. Which is the same incidence found in our study.

Kakarlapudi et al [19] noted that patients with diabetes were more commonly known to have hearing loss (13.1%) when comparing to the control group who were non healthy subjects without diabetes.

Wenget al [20] observed 67 patients with diabetes and found 44.8% among them had hearing loss which was profound. This is much lesser comparing to our study.

DURATION OF DIABETES:

In our study more number of patients with diabetes were found in 3-5 years group, about 48%, followed by less than 2 years duration 31%.

Comparing with, the duration of diabetes and hearing loss, more patients in the group 3-5 years had hearing loss, around 49%, followed by less than 2 years, around 32 years. Whereas only 20% patients were found to have hearing loss in 5-10 years group.

In study it is seen that the duration of diabetes is not a significant criteria for developing sensoineural hearing loss. The p value is <0.05, making it statically significant.

A study by Celik et al [21] noted that as diabetes duration raised to more than 15 years, the hearing loss incidence also increased. The influence on hearing loss was not significant after 15 years. This change was not observed in our study as cases in our study were all mostly found to be less than 15 years.

Overall severity of hearing in individual ear:

It is noted that in the right ear 95 cases had normal hearing, 84 cases had mild hearing loss, 15 had moderate and 3 cases had moderately severe and severe hearing loss respectively.

It is noted that in the left ear, 92 cases had normal hearing, 81 had minimal hearing loss, 16 had moderate loss 8 moderately severe, 2 with severe and 1 with profound hearing loss. Thus making it statically not significant ($p>0.05$). The above data was calculated taking the gender as consideration and compared the hearing. But no significant changes were noted in severity of hearing

Taking into consideration the duration of diabetes, it is noted that in right ear maximum of 58 (30%) cases had hearing loss among the group 3-5 years, among which 49 (25%) cases had mild hearing loss; 6 (3%) cases had moderate hearing loss and 3 cases (2%) had moderately severe loss, followed by 5-10 years group where 31 cases (16%) had hearing loss.

And in left ear it is noted that, maximum of 59 (30%) cases had hearing loss among the group 3-5 years, among which 44 (22%) cases had mild hearing loss; 8 (4%) cases had moderate hearing loss and 5 cases (3%) had moderately severe loss, followed by 5-10 years group where 32 cases (17%) had hearing loss.

Thus, we conclude that predominantly mild sensorineural hearing loss is noted individually in both ears. The p value is <0.01, making it statistically significant.

Overall severity of hearing loss:

In our study, 72% of individuals with diabetes were noted to have mild sensorineural hearing loss, followed by 20% with moderate sensorineural hearing loss, 3% and 4% had moderately severe and severe sensorineural hearing loss. Only 1% had profound hearing loss.

A study conducted by Rajendran et al showed a result similar to our study, The patients with diabetes reviled a high frequency loss which was bilateral and severity was from mild to moderate of sensorineural type and it was significant (73.3%) as compared to controls of similar age.

Whereas Weng et al [14] noted that in 67 patients with diabetes who were examined, 44.8% of them had hearing loss which was profound

Many studies were conducted to observe the pathogenesis of this sensorineural loss observed in diabetics. During this course many studies suggested that diabetes is a cause of hearing loss.

The probable mechanisms suggested were microangiopathy in the inner ear, neuropathy of cochlear nerve, or even a combination of both. Outer hair cell dysfunction and disruption of endolymphatic potential were noted in some studies. Some effects of diabetes on body tissue are thought to be correlating with the polyol pathway, where glucose is broken down to sorbitol.

Generally the accumulation of Sorbitol is observed in neuropathy as it causes a reduction in myo inositol levels which further leads to reduction in Na+ K+ ATPase activity [22].

Makishima and Tanaka [23] demonstrated atrophy of the spiral ganglion over basal lamina and the middle turns of cochlea were severe in individuals with diabetes along with hearing loss which was sensorineural type. It was also noted that in the 8th nerve changes were present in the myelin sheath, which denoted degeneration and fibrosis of perineurium

Jorgensen (1961) [24] in his study noted thickening in the vasa nervorum wall in the 8th nerve which lead to the development of acoustic neuropathy.

In the study done by Wackym and Linthicum (1986) [25] noted small microangopathic variations in the regions of endolymphatic sac, basilar membrane and the stria vascularis.

Van den Ouweland et al [26] observed mutational changes in the mitochondrial RNA. It was also observed that in a minor subset of patients, with diabetes inherited maternally showed some degree hearing loss which was sensorineural type.

Lisowska et al [27] in their study noted that in diabetic patients, there was abnormality in the function of outer hair cells in auditory brain stem responses.

Fukushima et al [28] proposed that patients with type 2 Diabetes had changes in cochlea, leading to atrophy of stria vascularis and basal turn which was significant and which could be the likelihood of hearing loss in these patients.

In our study, we conclude that after analyzing the audiograms of the diabetic patients, it is noted that they are more prone to develop mild to moderate sensorineural hearing loss. The duration of diabetes as well as sex of the individual were found not to have any effect on the incidence of hearing loss.

LIMITATIONS OF STUDY

1. The incidence of hearing loss in patients could have been more accurate with a larger group of study population. Only 200 diabetic patients were studied in study due to time and money constraints.

2. As the study was carried out in already diagnoised cases of diabetes, the correlation with HBA1c values which denotes the control of diabetes, could not be considered as there was a financial constraint, because the test had to be done as an added investigation in our study.
3. The occupations of the patients were not considered in the study which may influence the results of the audiogram this may be one of the drawback of the study.

CONCLUSIONS

1. The incidence of hearing loss among diabetics in our study is 55%.
2. More number of patients had senorineural hearing loss which was mild, followed by moderate hearing loss.
3. The incidence is greater in male patients.
4. According to our study, the peak incidence of hearing loss was seen among 50 -55 yrs age group of people.
5. In our study the duration of diabetes did not have an influence in developing sensorineural hearing loss.

BIBLIOGRAPHY

1] Dhingra P.L. Diseases of Ear, Nose and Throat. Elsevier. Fourth edition,2009.

2] Colin Mathers1, Andrew Smith2, Marisol Concha3; Global burden of hearing loss in the year 2000

3] World Health Organization. *World Health Report 2001. Mental health: New Understanding, New Hope.* Geneva, World Health Organization 2001

4] WHO Study Group. Diabetes mellitus. Technical Report Series 727. Geneva: World Health Organization, 1985.

5] Savage PJ, Bennion LJ, Bennett PH. Normalization of insulin and glucagon secreation in ketosis-resistant diabetes mellitus with prolonged diet therapy. J Clin Endocrinol Metab 1979;49:830-833.

6] Agner T, Damm P, Binder C. Remission in IDDM: prospective study of basal C- peptide and insulin dose in 268 consecutive patients. Diabetes Care 1987.

7] Gavin JR III, Alberti KGMM, Davidson MB, et al. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes care1997;20:1183-1197

8] Tanaka S, Kobayashi T, Momotsu T. A novel subtype of type 1 diabetes mellitus. NEngl J Med 2000;342:1835-1837.

9] Turner Rc, Cull CA, Frighi V, et al. Glycemic control with diet ,suphonyl urea, metformin, or insulin in patients with type 2 DM; Progressive requirement for multiple therapies. UK Prospective diabetes study group. JAMA 1999; 281 ; 2005-2012

10] King H, Rewers M. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. Diabetes care 1993;16:617-621.

11] Boyko EJ, de courten M, Zimmet PZ, et al. Features of the metabolic syndrome predict higher risk of diabetes and impaired glucose tolerance: a prospective study in Mauritius. Diabet Med 2003;20:915-920.

12] Gareth Williams, John C. Pickup. Text book of diabetes mellitus. Blackwell publishing Ltd, Oxford, Third edition, 2003.

13] Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF. Effects of type 2 diabetes mellitus on cochlear structure in humans.Arch Otolaryngol Head Neck Surg. 2006;132(9):934-8.

14] Maia CA, Campos CA. Diabetes mellitus as etiological factor of hearing loss. Rev Bras Otorrinolaringol.2005;71:208-214.

15] Taylor,I.G.,Irwin,J. Some audiological aspects of diabetes mellitus. J Laryngol Otol.1978;92:99-113

16] Rajendran S , Anandhalakshmi , Mythili B , Viswanatha Rao. Evaluation of the Incidence of Sensorineural hearing loss in Patients with Type 2 Diabetes Mellitus. Int J Biol Med Res. 2011; 2(4): 982 – 987

17] Cullen JR, Cinnamond MJ. Hearing loss in diabetics. J Laryngol Otol.1993;107(3):179-82.

18] Friedman SA, Schulman RH, Weiss S. Hearing and diabetic neuropathy. Arch Intern Med. 1975 ;135(4):573-6.

19] Kakarlapudi V, Sawyer R, Staecker H. The effect of diabetes on sensorineural hearing loss. Otol Neurotol. 2003 ;24(3):382-6.

20] Weng SF, Chen YS, Hsu CJ, Tseng FY. Clinical features of sudden sensorineural hearing loss in diabetic patients. Laryngoscope.2005;115(9):1676-80.

21] Celik O, Yalçin S, Celebi H, Oztürk A. Hearing loss in insulin dependent diabetes mellitus. Auris Nasus Larynx.1996;23:127-32.

22] Dennis L. Kasper, Anthony S. Fauci, Dan L.Longo, Eugene Braunwald, Stephen L. Hauser, J.Larry Jameson and Joseph

Loscalzo. Harrison's Principles of Internal Medicine. McGraw-Hill Companies Inc. United States of America.17th Ed.2008

23] Makashima,K.,Tanaka,K. Pathological changes of the inner ear and central auditory pathways in diabetics. Ann Otol Rhinol Laryngol.1971;80:218-288.

24] Jorgensen,M.B. The inner ear in diabetes mellitus. Arch Otolaryngol Head Neck Surg;1961;74:31-39.

25] Wackym,P.A.Linthicum,F.H.(1986)Diabetes mellitus and hearing loss: Clinical and histopathologic relationship. Am J.Otolaryngol.1986;7:176-182.

26] Van den Ouweland JM, Lemkes HH Gerbitz KD, Maassen JA. Maternally inherited diabetes and deafness (MIDD): A distinct subtype of diabetes associated with a mitochondrial tRNALeu(UUR) gene point mutation. Muscle Nerve. 1995; 3:S124-30

27] Lisowska G, Namyslowski G, Morawski K, Strojek K. Otoacoustic emissions and auditory brain stem responses in insulin dependent diabetic patients. Otolaryngol Pol. 2002;56(2):217-25.

28] Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF. Effects of type 2 diabetes mellitus on cochlear structure in humans. Arch Otolaryngol Head Neck Surg. 2006; 132(9)

29] Fangchao Ma, Orlando Gomez- Marin,David J. Lee, Thomas Balkany. Diabetes and hearing impairment in Mexican American adults: a population – based study. J Laryngol Otol. 1998;112: 835 – 839.

30] Gibbin KP, Davis CG. A hearing survey in diabetes mellitus. ClinOtolaryngol Allied Sci. 1981;6(5):345-50.

31] Austin DF, Konrad-Martin D, Griest S, McMillan GP, McDermott D, Fausti S.Diabetes related changes in hearing. Laryngoscope. 2009 ;119(9):1788- 96.

32] Sakuta H, Suzuki T, Yasuda H, Ito T. Type 2 diabetes and hearing loss in personnel of the Self-Defense Forces. Diabetes Res Clin Pract. 2007 ;75(2):229-34.

33] Davis A Hearing In Adults The Prevalence And Distribution Of Hearing Impairment And Reported Hearing Disability In The MRC Institute Of Hearing Research´s National Study Of Hearing. London 1994.

34] Reuben DBHL Prognostic Value Of Sensory Impairment In Older Persons 1999 Journal Of The American Geriatrics Society Vol 47(8), 930-935

35] Little P, Bridges A, Guragain R, Friedman D, Prasad R, Weir N Hearing Impairment and ear pathology in Nepal 1993 The Journal of Laryngology and Otology, Vol 107,395-400

36] Meecham E Audiolgy And Hearing Impairment: Improving The Quality Of Care 1999 Nursing Standard Vol 13(43), 42-46

37]. Nadol J Medical Progress: Hearing Loss N Engl J Med 1993 Vol 329(15), 1092-1102

38] Ramachandran A, Snehalatha C, Latha E, Vijay V, Viswanathan M. Rising prevalence of NIDDM in an urban population in India. *Diabetologia* 1997; *40* : 232-7.

39] Huizinga MM, Rothman RL. Addressing the diabetes pandemic: A comprehensive approach. Indian J Med Res 2006; 124 : 481-4.

40] Marine N, Vinik AI, Edelstein I, Jackson WPU. Diabetes, hyperglycaemia and glycosuria among Indians, Malays and Africans (Bantu) in Cape Town, Africa. Diabetes 1969;18:840-57.

41] Goldberg MD, Marine N, Ribeiro F, Campbell GD, Vinik Al, Jackson WPU. Prevalence of glycosuria and diabetes among Indians and Bantu. S Afr Med Y 1969;43 :733-8.

ABBREVIATIONS

PTA	-	Pure tone audiometery
Hz	-	Hertz
dB	-	Decibles
SPL	-	Sound Pressure Level
SRT	-	Speech Reception Threshold
HL	-	Hearing loss
SNHL	-	Sensorineural hearing loss
CHL	-	Conductive hearing loss
MODY	-	Maturity Onset Diabetes in Young
DM	-	Diabetes Mellitus
YLD	-	Years Lived with disability
FDA	-	Food & Drud Association

CONSENT FORM

I, Prathula Sivakumar am carrying out a study on the topic: A study on prevalence of hearing loss as a complication of Diabetes mellitus as part of my research project being carried out under the aegis of the Department of: ENT

My research guide is: Dr.Palaninathan

The justification for this study is: diabetic patients are more prone to complications of hyperglycaemia, as all body cells are exposed to high levels of plasma glucose. The organ of corti cells are important structures for hearing mechanism and turn out to be the potential target for damage, due to high glycemic levels, micro vascular compromise, their complex structure and arrangement. Thus screening of these patients at a higher risk of developing sensory neural hearing loss, will aid in early diagnosis and management

The objectives of this study are:

Primary Objective: To study the prevalence of hearing loss in diabetics.

Sample size: 200.

Study volunteers / participants are (specify population group & age group): Patients attending the outpatient services in department of ENT and diabetology in PSG IMSR between the age group 30 to 60 years.

Location: PSG IMSR.

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration): 2 minutes.

Data collected will be stored for a period of fifteen years. We will not use the data as part of another study.

Benefits from this study: Early diagnosis of hearing loss if present, early management.

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right to withdraw from the interview / study at anytime.** You have the freedom to withdraw from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date:

Witness:

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
1	45	2	2	5	2	13.3	41.6	1	3	2
2	36	1	2	1	1	26.6	26	2	2	1
3	47	2	1	2	1	30	30	2	2	1
4	55	3	2	20	4	40	31.6	2	2	1
5	60	3	1	15	4	55	55	3	3	1
6	43	2	1	3	2	28.3	30	2	2	1
7	58	3	1	3	2	40	56.6	2	4	1
8	55	3	2	5	2	38	33	2	2	1
9	46	2	2	4	2	28.3	35	2	2	1
10	37	1	2	10	3	20	25	1	1	0
11	48	2	1	1	1	33.3	33.3	2	2	1
12	55	3	1	6	3	28.3	28	2	2	1
13	60	3	1	5	2	35	32	2	2	1
14	50	2	1	10	3	36.6	40	2	2	1
15	41	2	1	3	2	12.2	15.5	1	1	0
16	39	1	2	5	2	25	20	1	1	0
17	57	3	2	5	2	38.3	30	2	2	1
18	43	2	2	10	3	25	24	1	1	0
19	46	2	1	6	3	83.3	60.7	5	4	3
20	59	3	2	6	3	35	35	2	2	1
21	39	1	2	2	1	28.3	28	2	2	1
22	56	3	1	6	3	35	31.6	2	2	1
23	50	2	1	8	3	30	40	2	2	1
24	36	1	1	1	1	21.6	25	1	1	0
25	60	3	1	3	2	38.3	35	2	2	1
26	60	3	1	4	2	35	33.3	2	2	1
27	45	2	1	25	4	30	26	2	2	1

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
28	57	3	2	1	1	36.6	30	2	2	1
29	38	1	2	5	2	20	23	1	1	0
30	58	3	1	4	2	68.3	61	4	4	1
31	45	2	2	10	3	28.3	31.6	2	2	1
32	48	2	1	5	2	50	48	3	3	1
33	47	2	1	10	3	26.6	35	2	2	1
34	54	3	2	20	4	25	20	1	1	0
35	43	2	1	1	1	28.3	30	2	2	1
36	39	1	1	1	1	43	32	3	2	3
37	43	2	2	5	2	15.2	13.1	1	1	0
38	58	3	2	1	1	20	21	1	1	0
39	46	2	2	5	2	14.5	15	1	1	0
40	47	2	2	1	1	21	23.5	1	1	0
41	55	3	2	10	3	30	27	2	2	1
42	59	3	2	5	2	21	23	1	1	0
43	31	1	2	2	1	16	15	1	1	0
44	52	3	1	3	2	23.2	25	1	1	0
45	52	3	1	5	2	56	85	4	5	2
46	45	2	2	2	1	23	25	1	1	0
47	44	2	2	1	1	15	17.6	1	1	0
48	55	3	1	10	3	26	25.3	2	2	1
49	55	3	1	1	1	23	20	1	1	0
50	49	2	1	3	2	20	70	1	4	2
51	56	3	1	1	1	40	35	2	2	1
52	58	3	2	8	3	25	23	1	1	0
53	51	3	1	2	1	31	30	2	2	1
54	48	2	1	3	2	30	28.6	2	2	1

						PTA				
S.NO	AGE	AGE	SEX	DURATION OF DM	DURATION OF DM	RIGHT	LEFT	RT HL	LT HL	HL
55	59	3	1	5	2	38	32	2	2	1
56	51	3	2	2	1	23	28	1	2	1
57	39	1	1	1	1	16	18	1	1	0
58	60	3	1	6	3	28	30.2	2	2	1
59	30	1	1	1	1	13.5	18	1	1	0
60	40	1	2	3	2	23	25	1	1	0
61	38	1	2	5	2	30	28	2	2	1
62	36	1	2	3	2	18	20	1	1	0
63	57	3	1	5	2	30	32	2	2	1
64	59	3	2	7	3	32	35	2	2	1
65	50	2	1	4	2	24	23	1	1	0
66	49	2	2	2	1	19	23	1	1	0
67	34	1	1	1	1	13.5	16	1	1	0
68	59	3	1	3	2	32	35	2	2	1
69	55	3	1	4	2	38	40	2	2	1
70	57	3	1	6	3	32	30	2	2	1
71	36	1	1	2	1	23	24	1	1	0
72	48	2	2	3	2	18	16	1	1	0
73	59	3	1	7	3	41	41.6	3	3	1
74	54	3	2	4	2	38	42	2	3	2
75	47	2	1	3	2	23	25	1	1	0
76	56	3	2	5	2	35	38.3	2	2	1
77	44	2	2	2	1	16	18	1	1	0
78	56	3	2	5	2	30	30	2	2	1
79	36	1	2	4	2	65	66.1	4	4	1
80	60	3	1	2	1	31.6	63	2	4	2
81	59	3	1	5	2	15.6	13.3	1	1	0

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
82	59	3	2	6	3	20	23	1	1	0
83	43	2	1	3	2	13.1	14.5	1	1	0
84	53	3	1	6	3	42	48	3	3	1
85	47	2	2	3	2	26	25.8	2	2	1
86	47	2	1	4	2	30.2	30	2	2	1
87	36	1	2	2	1	13.2	16.5	1	1	0
88	60	3	2	3	2	15	14.3	1	1	0
89	58	3	1	3	2	31.6	63.3	2	4	2
90	53	3	2	6	3	30	28	2	2	1
91	57	3	2	3	2	15	17	1	1	0
92	47	2	1	3	2	13	15	1	1	0
93	57	3	2	5	2	32	34	2	2	1
94	53	3	1	6	3	23	25	1	1	0
95	40	1	1	2	1	28.7	30	2	2	1
96	51	3	1	1	1	13.5	15	1	1	0
97	57	3	1	5	2	50.7	47	3	3	1
98	45	2	2	3	2	38	35	2	2	1
99	57	3	2	4	2	36	34	2	2	1
100	56	3	1	5	2	26	28.6	2	2	1
101	56	3	1	6	3	21	25	1	1	0
102	37	1	2	2	1	20	23	1	1	0
103	33	1	2	1	1	18	20	1	1	0
104	58	3	1	3	2	28	25	2	1	1
105	36	1	1	5	2	38	36	2	2	1
106	47	2	2	6	3	82	60.6	5	4	3
107	60	3	1	5	2	26	28	2	2	1
108	60	3	1	4	2	35	43	2	3	2

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
109	58	3	2	6	3	25	27.6	1	2	1
110	45	2	2	3	2	20.6	24	1	1	0
111	53	3	2	10	3	35	32.6	2	2	1
112	45	2	2	3	2	14	16	1	1	0
113	58	3	2	6	3	42	35	3	2	3
114	37	1	2	2	1	16	15	1	1	0
115	49	2	1	5	2	25.6	28	2	2	1
116	36	1	2	2	1	21	22.7	1	1	0
117	51	3	1	3	2	25.7	28	2	2	1
118	50	2	2	2	1	23	25	1	1	0
119	54	3	1	4	2	42	50	3	3	1
120	59	3	1	6	3	30	32	2	2	1
121	57	3	2	5	2	16	13	1	1	0
122	39	1	1	3	2	20	21	1	1	0
123	56	3	1	7	3	30	32.6	2	2	1
124	43	2	2	2	1	13	16	1	1	0
125	46	2	1	3	2	22.3	23	1	1	0
126	34	1	1	6	3	38.3	51	2	3	2
127	55	3	2	5	2	41.6	45	3	3	1
128	41	2	2	2	1	15	13	1	1	0
129	58	3	2	5	2	28	30	2	2	1
130	55	3	1	6	3	26	27	2	2	1
131	50	2	1	5	2	28	73.3	2	5	2
132	47	2	1	2	1	16	15	1	1	0
133	47	2	2	3	2	35	35	2	2	1
134	47	2	1	2	1	20	16	1	1	0
135	58	3	2	3	2	28	26	2	2	1

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
136	45	2	1	1	1	18	16	1	1	0
137	38	1	2	2	1	16	17	1	1	0
138	46	2	2	2	1	14	13	1	1	0
139	54	3	1	4	2	25.6	26	2	2	1
140	60	3	1	6	3	42	41.2	3	3	1
141	41	2	1	1	1	18	17	1	1	0
142	60	3	2	4	2	30	32	2	2	1
143	55	3	1	5	2	42	21.6	3	1	1
144	40	1	1	1	1	18	19	1	1	0
145	44	2	1	2	1	19	21	1	1	0
146	52	3	2	3	2	26	25.6	2	2	1
147	53	3	1	5	2	42	41	3	3	1
148	33	1	1	1	1	18	19	1	1	0
149	55	3	1	3	2	23	25	1	1	0
150	42	2	1	3	2	28	27	2	2	1
151	54	3	1	4	2	19	20	1	1	0
152	40	1	1	1	1	13	15	1	1	0
153	52	3	1	15	4	90	93	5	6	2
154	51	3	2	1	1	25	21	1	1	0
155	51	3	2	1	1	35	32	2	2	1
156	41	2	2	2	1	13.5	14	1	1	0
157	51	3	1	3	2	25.7	28	2	2	1
158	51	3	1	3	2	22	24	1	1	0
159	31	1	2	2	1	23	22	1	1	0
160	55	3	1	5	2	28	26	2	2	1
161	50	2	1	1	1	13	16	1	1	0
162	60	3	1	7	3	42	45.5	3	3	1

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
163	43	2	1	3	2	25.8	26	2	2	1
164	50	2	2	5	2	20.2	23	1	1	0
165	59	3	2	3	2	28	30	2	2	1
166	46	2	2	5	2	18	20	1	1	0
167	49	2	1	2	1	23	25	1	1	0
168	56	3	1	6	3	28	32	2	2	1
169	47	2	1	3	2	22	24	1	1	0
170	57	3	1	5	2	32	35	2	2	1
171	52	3	2	3	2	21	23	1	1	0
172	56	3	1	7	3	42	43.6	3	3	1
173	51	3	1	2	1	23	22	1	1	0
174	59	3	1	6	3	32	28	2	2	1
175	54	3	1	3	2	24	28.5	1	2	2
176	57	3	1	4	2	22	25	1	1	0
177	59	3	2	5	2	30	32.6	2	2	1
178	53	3	1	3	2	22	23	1	1	0
179	36	1	1	2	1	23	24	1	1	0
180	48	2	1	2	1	18	19	1	1	0
181	60	3	2	6	3	50	43	3	3	1
182	45	2	2	5	2	26	26	2	2	1
183	47	2	1	3	2	23	21	1	1	0
184	57	3	1	5	2	26	28	2	2	1
185	41	2	1	1	1	16	18	1	1	0
186	60	3	1	7	3	32	34	2	2	1
187	52	3	2	2	1	21	23	1	1	0
188	53	3	1	4	2	24	25	1	1	0
189	59	3	1	7	3	29	28	2	2	1

						PTA				
S.NO	**AGE**	**AGE**	**SEX**	**DURATION OF DM**	**DURATION OF DM**	**RIGHT**	**LEFT**	**RT HL**	**LT HL**	**HL**
190	32	1	1	1	1	16	18	1	1	0
191	49	2	1	2	1	23	25	1	1	0
192	43	2	2	2	1	18	18	1	1	0
193	48	2	2	4	2	28	29	2	2	1
194	43	2	1	2	1	13	16	1	1	0
195	50	2	1	4	2	19	23	1	1	0
196	43	2	2	3	2	26	25.8	2	2	1
197	42	2	2	2	1	23	24	1	1	0
198	44	2	2	4	2	23	25	1	1	0
199	36	1	2	2	1	15	17	1	1	0
200	55	3	2	5	2	28	28.9	2	2	1